THE CLINICAL EXAMINATION OF DISEASES OF THE LUNGS

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PREFACE

STUDENTS have a special difficulty in understanding clearly the various physical signs of pulmonary disease, in spite of the many excellent books at their disposal. This small work gives the outlines of our own teaching on the subject, and is published at the request of students and in the hope that it will be as useful to them as a similar work on diseases of the heart has been.

E. M. B.
A. R.

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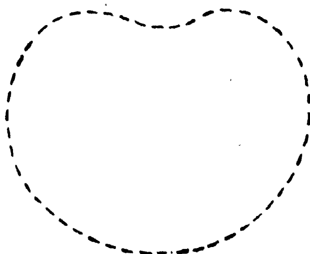
THE
CLINICAL EXAMINATION OF DISEASES OF THE LUNGS

I

PRELIMINARY CONSIDERATIONS

Anatomy and Physiology.

Shape of the chest.—The normal adult chest is of oval form, with the axillary regions somewhat flattened. The transverse diameter is greater than the antero-posterior in the ratio of about 7 to 5 or 4·5. In a baby the chest is nearer a circle in shape, approaching the adult shape in the early years of life.



Outline of Normal Chest.

The normal chest is ***bilaterally symmetrical*** in shape, size, and movement; the surface of the sternum is continuously level with that of the costal cartilages, and there is no actual hollow space under the collar-bones, but only a little natural depression between the pectoralis major and deltoid muscles, called the infraclavicular fossa.

Lungs.—The highest part of the lungs, in the majority of adults, is 1½ inches above the sternal end of the clavicle in the interval between the sternal and clavicular heads of the sterno-mastoid muscle.

The anterior edges of the lungs meet in the middle line at the junction of the manubrium with the body of the sternum. The edge of the *right lung* then runs vertically downwards in the middle line to the sixth rib, where it slopes downwards and outwards to the right, along the line of the sixth cartilage. The front edge of the *left lung* runs close to the right lung to the level of the fourth cartilage, when it turns off to the left to the apex of the heart in the fifth interspace, in the mid-clavicular line, 3½ inches from the mid-sternal line in the adult.

The lower border of both lungs follows a line running from the junction of the sixth costal cartilage with its rib horizontally round the chest, reaching the spine about the level of the eleventh dorsal vertebral spine.

The ***left lung*** has two lobes; the upper forms the apex of the lung and most of the anterior portion; the lower is in contact with the diaphragm, and forms all the posterior part except 2½ inches occupied by the upper lobe.

The ***right lung*** is shorter than the left, because of the right lobe of the liver rising up into the thorax. It has three lobes; the upper one is similar to the left, and the apex of the middle lobe is in the upper part of the axillary region. This is important for investigating tuberculosis of the apices.

Pleuræ.—The parietal pleuræ of both lungs practically meet just to the left of the middle line of the sternum, and run down to the level of the fourth costal

cartilage together; there the left pleura passes outwards to the left of the left margin of the sternum to the level of the xiphoid process, whence it runs along the line of the sixth costal cartilage to the junction of the eighth rib with its cartilage. The right parietal pleura continues down the mid-line of the sternum to the xiphoid process, where it is reflected along the line of the seventh costal cartilage to the junction of the eighth rib with its cartilage. From this last point the pleuræ of both lungs run downwards and outwards and cross the twelfth rib about its mid-point, and at the level of the first lumbar vertebra.

Posteriorly, the line of the pleuræ for all practical purposes is down that of the sides of the dorsal vertebræ. It is sometimes stated that the left pleural sac reaches a lower level than the right, but this is doubtful in fact and immaterial in practice.

The trachea divides opposite the junction of the manubrium and the gladiolus sterni in front, and the fourth dorsal vertebra behind.

Respiratory movements.—The normal movements of respiration are those of inspiration and expiration.

Inspiration.—(*a*) Each succeeding rib as far as the seventh has a larger curve than its predecessor, and when raised *increases the transverse diameter* of the chest.

(*b*) Owing to the "twist" in the ribs, when they are raised as a lever the sternum is pushed forward, and *increases the antero-posterior diameter* accordingly.

(*c*) The contraction of the diaphragm reduces the concavity directed into the thorax—that is, it increases the vertical diameter of the thorax. This causes compression of the abdominal organs, which is compensated for

by (1) outward movement of the lower ribs ; (2) elasticity of the abdominal wall.

Expiration, in normal breathing, is brought about by the cessation of the inspiratory muscular effort, when the weight of the thorax and the elastic recoil of the lung tissue are sufficient to expel the inspired air. The abdominal wall muscles help materially by pulling down the lower ribs.

In women normal breathing is more costal and less diaphragmatic in type than it is in men.

Muscles of forced inspiration.—When there is obstruction to the entry of air into the alveoli of the lungs, and the ordinary muscles are insufficient to expand the chest, additional muscles come to their aid, and their action is plainly visible. These are muscles attached to or arising from the thorax, and attached to or arising from the base of the skull, the spinal column, and the humerus. The one whose activity is most easily seen is the sterno-mastoid.

Muscles of forced expiration.—All the muscles of the abdominal wall, with their attachments to the lower ribs, act in forced expiration, as anyone who has a troublesome cough will feel for himself.

The action of the muscles of inspiration is to increase the cubic capacity of the chest, by raising the ribs and sternum, and depressing the arch of the diaphragm. This results in the production of considerable *negative pressure within the chest*, and external positive pressure on the chest walls, until air has rushed into the lungs through the respiratory passages, and equalized the two pressures.

The normal chest wall can withstand the strain on it

and keep its shape when there is no obstruction to the free entry of air into the alveolar spaces of the lungs.

Movement of the chest in respiration.—In the normal person the chest moves equally well on both sides during respiration, and in quiet respiration it increases about 1 inch on the average in circumference at the level of the nipples. The difference between the measurement round the chest in deep inspiration and deep expiration is about 2 to 3 inches, and in well-made chests of people who take a good deal of exercise may be 4 to 4½ inches. These measurements are looked upon as being a good indication of a person's power of resistance to lung diseases, especially to phthisis. The expansion in a flat chest is much less than this, and it is so also in people with emphysema.

It is a curious fact that some people with healthy chests are unable to make deep in and out breathing movements to enable the physician to get the requisite measurements. They take a deep breath, and cannot let it out properly. The result is a difference in measurement of about 1 inch, or even only ½ inch, which has to be explained in any medical report. Corpulent people with protuberant abdomens are specially unable to expand their chests decently—½ inch being a difference not infrequently measured.

Insurance companies are particular in asking for the above measurements, for the reasons given, and some also ask as a confirmatory check if the air enters well into the bases of the lungs—that is, is the respiratory sound over the bases as clearly heard as it should be ?

The duration of the movements of inspiration and expiration is nearly but not quite equal, expiration being slightly the longer of the two. Normally, breath-

ing goes on in any of the usual positions of the body, and is quite easy when the individual lies flat on his back in bed, or on either side. Healthy people can breathe and sleep on either side or on the back, it being merely a matter of habit which position is chosen. The importance of this normal condition of respiration will be seen when we speak of disease.

The number of respirations in quiet normal breathing is about 18 per minute, or approximately 1 to every 4 pulse-beats. The stimulation for the respiratory movements arises in the centre in the medulla, and varies directly with the amount of carbonic and other acids in the blood. The number of respirations increases when there are additional demands for oxygenation of the blood, as, for instance, during or after exercise, when the pulse-rate also increases, but the ratio of 1 to 4 remains about the same.

In disease the number of respirations per minute is increased by various causes.

II

ROUTINE EXAMINATION

SUBJECTIVE SYMPTOMS.

SUBJECTIVE symptoms are those manifestations of disease which the patient is conscious of himself, and complains about. In some illnesses the patient's own statement of its presence is the only evidence the physician has of the symptom referred to. In other illnesses the physician can see for himself that the condition complained of is present—*e.g.*, shortness of breath—and this is called an *objective* sign or symptom.

The most common subjective symptoms in diseases of the lungs are pain, shortness or tightness of breath, and cough; general weakness and sweating and loss of flesh may also be complained of.

Pain in pulmonary disease arises from two conditions:

(1) ***In acute inflammation of the air passages,*** larynx, trachea, or bronchi, the mucous membrane at first is red and inflamed and "raw." There is very little secretion, but the irritation of the rawness causes a cough which is painful and only relieved when secretion becomes free.

(2) ***In inflammation of the pleura*** the pain is of a stabbing character, "like a knife sticking in" or "a stitch," and is made worse by breathing or coughing. It is felt over the site of the inflammation.

When the diaphragmatic area of the pleura is in-

flamed, the pain is referred to the skin over the shoulder-joint.

The pain of pleurisy is often accompanied by a short, dry cough. In inflammation of the lungs any pain which may be felt is pleuritic in origin.

In chronic dry pleurisy an extensive friction may sometimes be heard or felt by the observer with practically *no pain* to the patient.

Diagnosis.—The pain of pleurisy must be differentiated from that of intercostal *neuralgia;* from that which is premonitory to an attack of intercostal *herpes zoster;* and from *rheumatism* of the chest muscles. Pain in all of these conditions is different in character from pleurisy pain, being more of a constant aching, burning pain, and not of a stabbing character, not influenced so much by the respiratory movements.

Referred pain—***Shoulder pain.***—When the diaphragmatic pleura is inflamed, pain is often referred to the skin over the corresponding shoulder-joint supplied by the cutaneous branches of the fourth cervical nerve, which sends muscular fibres by the phrenic nerve to the diaphragm. This is a very characteristic symptom, the patient placing his hand over the top of the shoulder-joint to indicate the seat of the pain. It is not uncommon as an early sign in pneumonia involving the base of the lung.

Abdominal pain.—This is not uncommonly complained of in pleurisy in children, when the diaphragmatic pleura is inflamed, and is a misleading symptom unless its occurrence is known.

Angina Pectoris.—The pain of true angina pectoris, the result of weakness of the heart muscle brought on

by narrowing of the coronary arteries or overstrain, is so characteristic that it is hardly likely ever to be confused with pain in the chest due to pulmonary disease. It consists essentially of pain in the chest and a sense of impending death, as if the chest were gripped in a vice. The pain begins over the sternum, often at its lower part, and spreads to the præcordia up the neck and down the inner side of the left arm, or both arms.

Shortness or tightness of breath is complained of when any considerable portion of the alveolar tissue of the lungs is put out of action by inflammatory processes; by fluid in the pleural cavity; by the invasion of new growth; by pressure on a bronchus; and by narrowing of the lumen of the air passages in asthma. The air hunger is so great in asthma that sometimes the patient opens the window and leans out "to get as much air as he can."

In emphysema, with the thorax in a constant state of full inspiration and very little tidal air entering the chronically expanded lungs with each inspiration, there is a definite feeling of shortness of breath. Any coincident heart muscle weakness will make breathing more difficult.

Cough of varying character, with or without expectoration, is generally complained of (see special section).

Exhaustion is often a very important symptom in early stages of phthisis. If a patient complains of always feeling tired, or of becoming exhausted and good for nothing early in the day, and without any exertion to cause it, this disease must be suspected. Careful examination may reveal no physical signs, or

only very slight ones. Such a case must be watched and other means of diagnosis employed, especially the behaviour of the temperature after exercise and the examination of the sputum for tubercle bacilli.

This feeling of exhaustion is not the same as breathlessness or dyspnœa, but a general mental and bodily feeling of tiredness or lassitude.

The body-weight may be an important guide to the diagnosis of phthisis. In many cases a definite history of gradually decreasing weight is the first indication of the onset of tuberculosis, and for this reason it is of the greatest importance to obtain accurate information on the point, and to inquire for any other possible cause of loss of weight.

If there is a family history of pulmonary tuberculosis, members below the average weight for their height and their age must be looked upon as more likely to develop phthisis than those of average or above average weight, because of their inability to assimilate food properly. An increase in weight is a favourable sign in the prognosis for patients who have or are threatened with consumption.

Night sweating may be an early symptom complained of by people suffering from, or threatened with, pulmonary tuberculosis. The sweats may be profuse and distressing, waking the patient up, and requiring a change of night garments. Night sweats occur in some other pyrexial conditions in adults, such as influenza and septicæmia, and in rickets in children.

Objective Signs.

Objective signs are those physical signs which can be made out by the physician himself. After hearing and making a note of the subjective symptoms, or what the

patient complains of, the examination for objective symptoms must be carried out in a systematic way. It is always best to begin by feeling the pulse and ascertaining the temperature; the chest should then be examined by inspection, palpation, and percussion and auscultation, and the sputum must be examined.

Pulse.—The pulse may afford us some information of diagnostic value in pulmonary disease, apart from the quickening incident to any rise of temperature.

In ***lobar pneumonia*** the pulse-rate increases with any rise of temperature, and in severe cases becomes quicker still from toxæmic causes. There is nothing of diagnostic value in this fact alone, but when the increased pulse-rate is compared with the increased respiration-rate we get some useful information.

In pneumonia the increase in the number of respirations is always proportionately greater than the increase in the number of heart-beats, and instead of being 1 to 4, as in normal conditions, it becomes 1 to 3, or even 1 to 2.

Pulmonary tuberculosis.—In early stages of this disease the pulse-rate may be rapid, 120 or more, without the patient knowing of it or complaining of breathlessness, even on walking some distance. A sense of exhaustion or of fatigue early in the day and without any cause may be noticed, but no rapid action of the heart is complained of.

N.B.—A persisting rapid pulse may be one of the earliest warnings of the onset of pulmonary tuberculosis.

When the disease is advanced a quick pulse is common, without there being any actual disease of the heart.

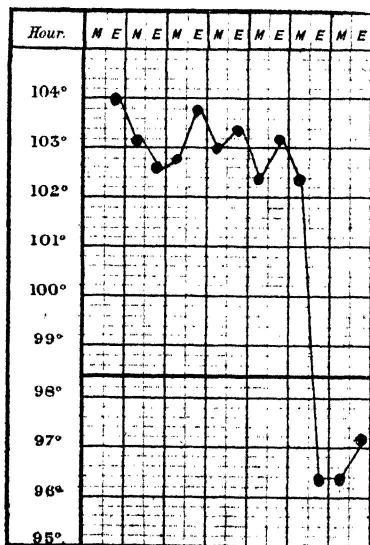
Pleuritic effusion.—If the heart is displaced materially by pleuritic effusion or any other cause, it will beat

feebly and quickly, and this is one indication for removal of pleuritic effusion.

In ***chronic bronchitis*** and ***emphysema*** it is often astonishingly slow and regular. The muscle has responded to the gradual demands on it, and does its work well for years. Muscle failure may eventually develop, beginning on the right side.

Asthma.—In the paroxysm of asthma the pulse may be quick, weak, small, and irregular.

Mediastinitis.—In chronic inflammatory fibrosis of the mediastinum the pulse is smaller during inspiration than during expiration (*pulsus paradoxus*).



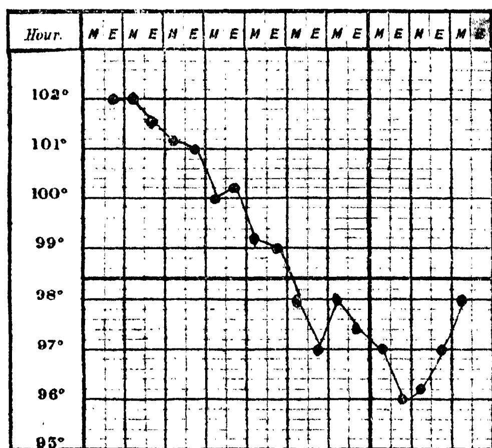
LOBAR PNEUMONIA: RESOLUTION BY CRISIS.

Temperature is a useful guide to the diagnosis of pulmonary affections, both in its mode of onset, its continuance, its duration, and its termination. Full information on this head will be found in text-books of medicine. There are, however, some points of diagnostic importance and interest which may be briefly referred to here.

Acute pneumonia.—In a typical case of acute lobar pneumonia, produced by the pneumococcus, there is a sudden onset, often with a rigor or shiver, the temperature rising to 103° or 104° in three or four hours

It remains high, 103° to 105°, for three or four days, then falls a couple of degrees in the morning, rises again at night, to fall below normal as suddenly as it rose, about the sixth or seventh day after the onset of the illness. The fall is by *crisis*, and this may be a fall of 6° or 7° in twelve hours. It remains subnormal, between 97° or 98°, for a week or so, gradually rising to normal as convalescence is well established. In 75 per cent. of the cases the crisis occurs on or before the seventh day; in 18 per cent. on the fifth day, 14 per cent. on the sixth, and 24 per cent. on the seventh day.

Resolution by lysis.—Instead of ceasing by crisis, the temperature, in about one-third of cases which recover,

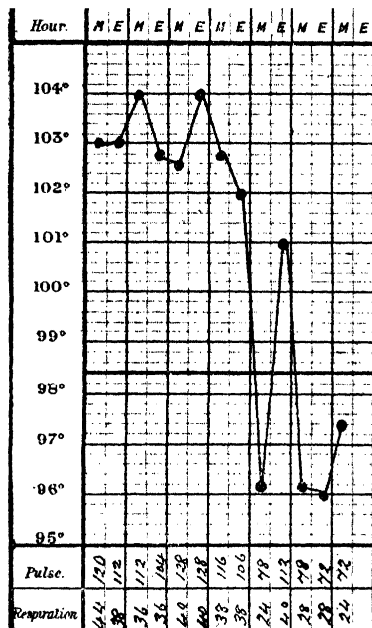


LOBAR PNEUMONIA: RESOLUTION BY LYSIS.

comes down gradually—that is, by *lysis*—to normal, occupying over this one and a half to three days.

Delayed resolution. — In other cases, again, the temperature may come down by lysis, but keeps between 101° or 101·5° in the evenings and 99° in the mornings

for one, two, or three weeks, gradually, however, coming down to the normal. In these cases there is delayed resolution—that is, the inflammatory material is not absorbed as rapidly as in the typical attack. The difficulty in this condition is to decide whether there is any complication, chiefly the formation of an *empyema*, and it is often necessary to make an exploratory puncture to settle the question. The possibility of *typhoid fever* being present must also be remembered, and the Widal test be done. See also Tuberculosis, p. 16.



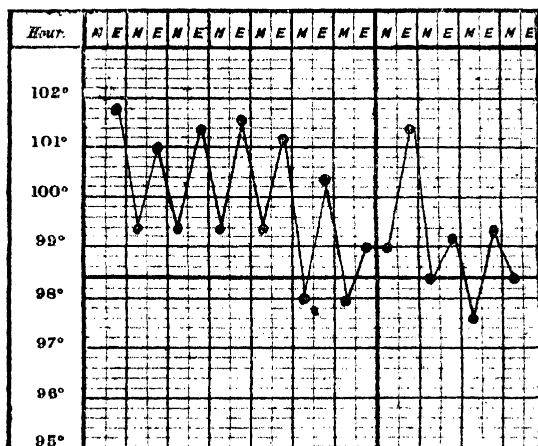
Lobar Pneumonia: Resolution by Pseudocrisis and Crisis.

Pseudocrisis.— In a certain percentage of cases of typical lobar pneumonia, about the third or fourth day of the disease, the temperature falls several degrees in a few hours to below normal, like in the crisis. It rises again, however, with equal rapidity, until it is as high as ever. This false crisis or pseudocrisis occurs about thirty-six hours before the true crisis, and a knowledge of its possible appearance is desirable, in order to warn the friends that such a fall is only temporary, and that the temperature will probably be as high as ever again in the evening.

Hyperpyrexia.—In a few cases of acute pneumonia, instead of the temperature falling it rises progressively, and the patient dies with it at 110° or higher.

Broncho-pneumonia, Septic pneumonia. — Many cases of pneumonia, especially those caused by the influenza bacillus or septic organisms, depart very considerably from the lobar pneumonia type, beginning more gradually, lasting longer, and ending more slowly, with accompanying alterations in the temperature.

Empyema.—When the temperature of an attack of acute pneumonia has come down to normal by crisis and

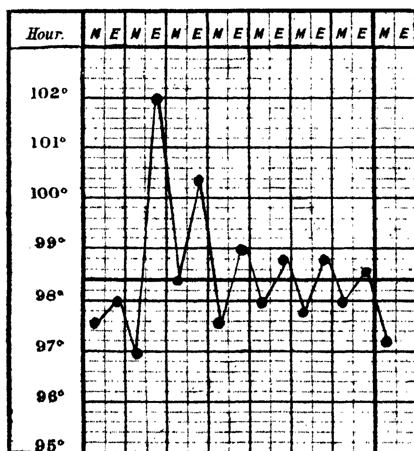


EMPYEMA.

ordinary lysis, it ought to remain there if the case is progressing favourably. If it begins to rise to 100° or 101° or more in the evenings, to fall to normal again in the morning, and continues doing so for a few days, it is very probable that an empyema has developed. Exploration by needle is then necessary.

Pulmonary tuberculosis.—The temperature in pulmonary tuberculosis is a toxæmic temperature with morning remissions and evening rises, caused by the products of activity of the tubercle bacilli and complicating septic organisms. It varies in range with the severity of the illness. Its presence must be suspected when there is *delayed resolution* in pneumonia.

An *evening rise* of temperature after the day's work may be an early symptom of tuberculosis. When a rise of temperature is suspected, the thermometer should be used every four hours during the day, because in tuberculosis the temperature may be highest, not in



ACUTE BRONCHITIS OR ACUTE PLEURISY.

the evening, as is usual, but at mid-day or even earlier.

Acute bronchitis.—The onset of the illness is gradual, the temperature rising in the course of twenty-four or

thirty-six hours to 101° or 103°, according to the severity of the attack. The patient may have a shivering feeling, like that of a severe cold, but there is no rigor. The illness in its milder forms is often spoken of as a "cold on the chest." The temperature keeps high for two, three, or more days, according to the severity of the illness, and then gradually subsides. In children the smaller bronchi are often involved, and this increases the amount of fever and prolongs the illness.

Acute pleurisy.—The temperature in acute pleurisy is practically like that in acute bronchitis, and a diagnosis can easily be made by the physical signs present, especially the presence of a friction rub, and later of effusion and displacement of the heart. The temperature may be elevated for a few days only, or it may continue for two or three weeks, gradually becoming normal. It is not at all like an acute pneumonia temperature.

Empyema.—If the serous effusion turns purulent the daily range of temperature is generally more extensive, and of the septicæmic type. The presence of pus must then be suspected, and a puncture with an exploratory syringe made. Empyema may develop in a few days from acute pleurisy.

Tuberculous pleurisy.—Persistence of temperature for some weeks in pleurisy, with or without any suppuration, must suggest the probability of a tuberculous origin of the disease, which happens in more than half the cases of pleurisy.

The effect of exercise on the temperature may be a useful means of diagnosis in early pulmonary tuberculosis. In the normal person it may rise ½° to 1° after a round of golf or a two or three miles walk, coming

down to normal again in the course of half an hour. In tuberculosis the temperature, under similar conditions, will often rise higher, to 99·6° or 100° F., and keep up longer. Therefore it is advisable to tell the suspected person to take his temperature before starting out on the test exercise, and then to take it afterwards every quarter of an hour for two hours. If the temperature remains raised for more than half an hour to one hour, it is probable that early tuberculosis is present.

III

INSPECTION

Changes in the Shape of the Chest.

The shape of the chest may be very much altered in disease from that of a bilaterally symmetrical oval, flattened somewhat in the axillary regions, and the alteration may affect the whole of the chest or part of it only.

A life-size outline of the chest can be made by the *cyrtometer,* which consists of two strips of lead "ribbon" loosely connected together at one end by a piece of flexible leather. The junction is applied to the spine of a dorsal vertebra, and each of the lead ribbons is then moulded to the shape of one side of the chest in a horizontal line, the two ends meeting over the sternum. The apparatus is then taken from the chest, and an outline of its form traced on paper.

For the chest to maintain its normal shape the following conditions are necessary :

(1) The spinal column and its musculature must be normal.

(2) The ribs, sternum, and costal cartilages must be normal in structure, so as to be able to withstand the suction pressure put on them during inspiration.

(3) There must be free entry of air into the alveolar tissue through the respiratory passages—nose, larynx bronchial tubes, and smaller air passages.

(4) The lungs must retain their normal structure, and be able—

(*a*) To expand fully and freely during inspiration; and

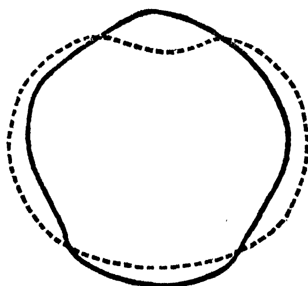
(*b*) To return to their normal size afterwards.

(5) The parietal and visceral layers of the pleura must be normal and move freely over each other, and not be adherent.

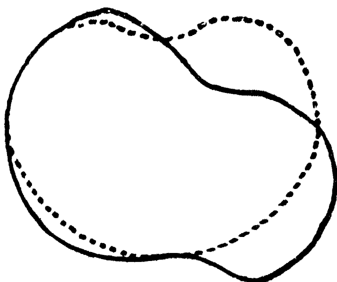
Departure from the normal in any one or more of these conditions will result in various alterations in the shape of the chest.

Both sides of the chest affected.—

(1) ***Changes in the spinal column,*** whether due to disease of the bones or to muscular atrophy, or carrying of heavy weights on one side in childhood, will affect the shape of the chest according to whether the abnormal



KYPHOSIS.



SCOLIOSIS.

Dotted line is the normal.

curvature of the spine is in an antero-posterior or lateral direction.

With *angular curvature* (kyphosis) the deformity of the chest is bilaterally symmetrical, the antero-posterior diameter being increased.

When there is a *lateral curvature* (scoliosis) of the spine, one side of the chest is more capacious and prominent than the other. One side is abnormally large and the other abnormally flat.

(2) ***Changes in the ribs, cartilages, and sternum,*** frequently occur in children as the result of *rickets*. The shaft of the rib is weakened, and its ability to resist pressure lessened. Its junction with its costal cartilage is enlarged and irregularly ossified; this leads to their *nodular enlargement*, and the formation of what is called the "rickets rosary." These nodules are easily seen in children, but are not so well marked in adult life, when the bones have reached their full size.

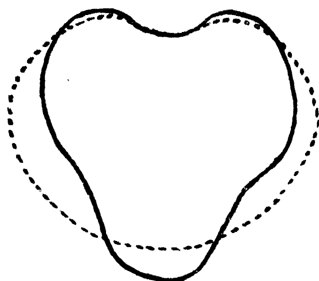
(3) ***Effects of suction.***—If there is obstruction to the free entry of air into the lungs, parts even of the normal bony thorax are unable to resist the pressure and are sucked in, but such changes are most marked when there is weakness of bony structure present as well. As the result of these processes, various deformities of the thoracic walls arise.

Parasternal groove.—The ribs and costal cartilages may give way, and a groove is formed down the line of the costal cartilages on both sides of the sternum.

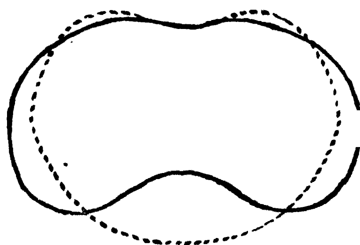
Pigeon chest.—The ribs may be distorted at the angle and flattened on their anterior half, and then the sternum becomes unusually prominent, thus forming the pigeon chest.

Depressed sternum.—The lower half of the sternum may be permanently depressed, leading to a hollow in the middle line of the chest—that is, to a depressed sternum. A similar condition is met with in *cobblers*, who are constantly pressing with their sternum against their work.

A *transverse groove* around the lower part of the chest along the attachment of the diaphragm, known as



PIGEON CHEST.



DEPRESSED STERNUM.

"Harrison's sulcus," may form. This is often present after whooping-cough, and also in clerks or students who habitually stoop over their desks, or in people who hold themselves badly.

The obstruction to the free entry of air into the lungs in these cases is generally caused by—

(1) *Adenoids* blocking the nasal respiratory passages; or

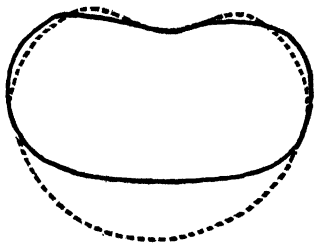
(2) Spasm of the larynx in whooping-cough.

(3) *Chronic bronchitis* or broncho-pneumonia, commonly met with in rickets, narrowing the lumen or free way of the air passages.

The ribs in these cases may not be abnormally weak, but as there is obstruction to the free entry of air into the lungs to counteract the negative pressure of respiration they are sucked in with every inspiration and eventually deformed.

(4) The obstruction to the free entry of air into the spongy portion of the lungs arises from the causes just mentioned.

The flat, pterygoid, alar, phthinoid chest.—One common bilaterally symmetrical abnormality is met with when the chest is badly developed, hollow, or flat under both collar-bones. The first intercostal space is distinctly farther back than normal, and the antero-posterior diameter of the thorax is abnormally shallow.



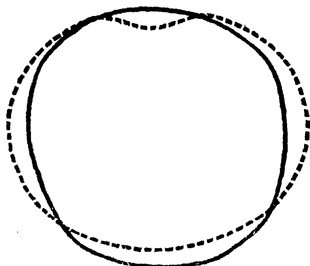
FLAT CHEST: BILATERAL DEFORMITY.

In typical cases one shoulder-blade stands out prominently like a wing—hence its name, alar or pterygoid. It is met with in people likely to develop phthisis—hence its name "phthinoid." Such a chest expands badly on inspiration.

Many people, especially those of sedentary life, without any tendency to phthisis, who "carry themselves badly" become flat-chested; they also often have a groove across the lowest ribs ("Harrison's sulcus," *q.v.*).

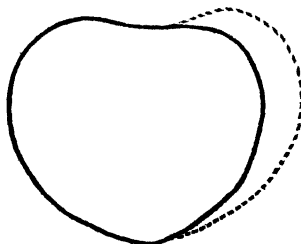
The long barrel-shaped chest.—Whilst the normal chest is roughly oval in shape throughout its whole length, it is not uncommon to meet in insurance work, or examining recruits, or in a hospital, people with no disease, and certainly with no lung disease, with a long barrel-shaped chest. This form of chest is longer proportionately to its width than the normal chest, and is rounder, especially in its lower half, than the normal chest. It is well developed under the collar-bones, and by no means flat and similar to the flat phthisical chest. The people with it have small bones generally. There is no emphysema, and no evidence of a tendency to phthisis, and it is not the result of tight-lacing.

Bilateral enlargement of the chest.—The lungs may increase in size from distension of the alveolar tissue, as in emphysema; both sides of the chest are affected, and the so-called *barrel-shaped chest*, with increased antero-posterior diameter, results.



EMPHYSEMA OR BARREL CHEST.

The dotted line is the normal.



UNILATERAL COLLAPSE.

The dotted line is the normal on the collapsed side

One side or part of one side affected.—

Flattening of one side.—One lung, or part of one lung, may be unable to expand freely for several reasons, with the result that it does not fill the thorax about it, and the walls of the chest fall in or become flattened.

(1) *Atelectasis or collapse*—that is, inability of the lung to expand after the removal of a pleural effusion which has been compressing it.

(2) *Fibrosis.*—Atrophy of the spongy tissue or increase of interalveolar tissue, forming pulmonary fibrosis, resulting from inflammatory processes. The deformity is generally one-sided, and affects the upper lobe, and is seen best under the collar-bone.

(3) *Pleural adhesion and thickening* lead to flattening of the chest over the site of the lesion, generally the

lower part of the chest. The thickened and adherent pleuræ draw the ribs nearer together, obliterate the intercostal spaces, flatten the chest, and impede the chest movements considerably.

(4) *Excision of ribs.*—Removal of part of one or more ribs for the relief of empyema leads to a marked flattening of part of one side of the chest.

Unilateral enlargement is met with when one lung expands to compensate for collapse or disuse of the other lung. In this case neither side may be normal, but one side may be abnormally full and the other abnormally flat. It is also met with in *pneumothorax.*

The condition of development of a chest under the collar-bones is always asked for in life insurance reports, any flattening, especially if unilateral, being looked upon with great suspicion as indicating a tendency to phthisis or as an evidence of some abnormality of the lung.

Local bulging.—Some of the intercostal spaces may be unusually prominent and convex over a large pleural effusion or an empyema. An empyema may point through an intercostal space, and make a distinct swelling. In one boy it pointed in the fourth right intercostal space, and looked like a small breast.

Other causes of local bulging.—A large diseased *heart* in children will cause a distinct bulging on the left side over the præcordia.

Aneurysm.—A very important and common cause of local bulging of the anterior chest wall, and less commonly of the posterior wall, is aneurysm of the arch of the aorta, especially the first and second portions. The pulsation is generally easily visible in a good light, if not from in front of the patient, then by

taking a tangential view of the chest. Such a condition does not concern us, unless it causes secondary pressure symptoms on the lungs or bronchi.

Disease of bone.—Tuberculous and gummatous disease of the bones, ribs, sternum, and vertebræ, are the next common causes of local swellings, and malignant disease also may affect a rib or ribs.

Pulsation due to pulmonary or pleural disease is very rare. An empyema on the left side, limited by adhesions and in contact with the pericardium, may transmit the pulsations to the surface, where they may be seen. It is a rare condition, however.

The position of the heart in the thorax must be carefully ascertained.

The cardiac impulse, if not visible, should be felt for, as the position of the heart in the thorax may be a very important indication of pulmonary or pleural diseases. When it is to be felt, the exact position of the left side, as indicated by the true apex-beat, should be felt for by one finger in an intercostal space, as advised in the book on the "Physical Signs and Symptoms of Heart Disease," by one of us. [E. M. B.]

The **condition of the heart** must be ascertained accurately in any pulmonary disease, for the heart is often a very important contributory cause of pulmonary disease.

Obstruction of the mitral valve and cardiac muscle failure of the right ventricle are specially to be mentioned in this connection.

Displacement of the heart in pulmonary disease may be caused in one of two ways. It may be—

(1) ***Pushed out of place*** to the right or to the left by effusions in one or other of the pleural cavities, by new

growths of the pleura or lungs, or by air in a pleural cavity ; or

(2) ***Pulled out of place,*** generally either upwards and outwards, by shrinking or collapse of part or whole of a lung, resulting from tuberculous disease.

Dextrocardia or congenital development of the heart on the right side will be accompanied by similar transposition of the lungs, the right then having only two lobes, whilst the left will have three.

Displacement of the heart from causes not of pulmonary origin—heart disease, aneurysm, increase of contents of abdomen—must be eliminated.

General Inspection.

With a knowledge of the general appearance of a healthy person in mind, a careful inspection of a patient may yield many signs of pulmonary disease.

The appearance and expression of the face, the general appearance and bearing, the shape of the chest, the nature of the breathing, and the presence or absence of a cough, should all be noted.

The face.—In many cases of pulmonary disease there is nothing unusual to be seen in the face, but in some important diseases there are signs which are of much diagnostic value. The chief points to be noted are the presence or absence of flushed cheeks, indicating a raised temperature, cyanosis, and any *movement of the alæ nasi* during respiration. The condition of the *pupils* should be noted, as it should be in all diseases, for inequality of them may be present, and be due to unilateral phthisis, pleurisy, and other pulmonary or intrathoracic lesions, including diseases of the thoracic aorta (aneurysm), pressing on the sympathetic nerve.

Evidence of *dyspnœa* or difficulty of breathing can be seen by the erect position in bed, in the face by the open mouth and distended alæ nasi, and in the neck by the action of the sterno-mastoid muscles (forced inspiration), and by the general look of air hunger.

Painful sensations and wincing, caused by acute pleurisy of whatever origin, are revealed in the face; they may also be seen in the early stages of acute inflammation of the large respiratory passages, larynx, trachea, or bronchi, when there is a short, painful, irritating, persistent cough with little expectoration.

Short, quick, shallow breathing is also very characteristic in acute plastic pleurisy before effusion develops.

In acute pneumonia a typical appearance is the flushed face of high temperature, the alæ nasi working, quickened respiration, short, snatchy breathing, and evidence of pain if there is pleurisy; and in many cases herpes zoster about one or other angle of the mouth.

Herpes labialis, with its very characteristic clump of vesicles or purulent crusts, is more commonly met with in acute pneumonia than any other febrile disease. It is frequently seen in this disease, especially in children, and will often give the experienced observer a clue to the diagnosis of the condition.

Chronic bronchitis and emphysema can often be suspected by a glance at the face, which has an ashy, more or less dirty bluish, or in later stages a *cyanotic,* appearance, with more of a true cyanosis in the lips, the lobes of the ears, and tip of the nose, which is caused by imperfect oxygenation of the blood. The same appearance may be seen transiently in severe attacks of asthma.

Phthisis.—In its early stages some patients may look "the picture of health." Other patients may look

anæmic, "delicate," and have long eyelashes, sunken but bright eyes, with pearly conjunctivæ, and dark rings under their eyes and down on the skin.

When the disease is advanced, the patient looks ill and emaciated, with sunken cheeks and prominent bones. If any temperature be present, there may be a hectic flush on the cheeks, and if the extent of lung affected is considerable this flush has a slightly cyanotic tint, and there is cyanosis of the lips.

In *acute* or *galloping phthisis*—phthisis florida—the skin is pale, with the flushed cheeks of a septic temperature.

Asthma.—During a paroxysm the expression is typically that of marked anxiety.

Neck.—When ***dyspnœa*** is present and the muscles of forced inspiration are acting, the *sterno-mastoid* muscles will be prominent and their contraction easily seen. When there is *venous obstruction*, from pressure on the large venous trunks by new growth in the chest, the veins of the neck are distended, and there may be some cyanosis and œdema of the neck and head and upper extremities. A distended jugular vein in such a condition will fill from above downwards, which is the opposite to what happens when the vein is distended from cardiac back-pressure causes.

Clubbed fingers and toes.—By clubbing is meant a club-like or bulbous swelling, and often flattening, of the terminal phalanges of the fingers and, to a less extent, of the toes, the soft tissues and not the bone being involved. Very often, too, there is marked congestion or cyanosis as well as clubbing, especially in congenital heart disease. Early conditions of it hardly bear out this description, and have to be looked for de-

liberately. The commonest to find is a marked exaggeration of the convexity of the nail, producing a resemblance to a puffin's or parrot's beak. Always look at the finger-tips when feeling the pulse of a case of pulmonary or heart disease.

Clubbing of the fingers or toes is met with most commonly in phthisis, in bronchiectasis, and in chronic bronchitis with emphysema. It is also met with in fibroid conditions of the lung, and in collapse from pressure of fluid in the pleural cavity.

The explanation of the change is difficult to find. It is met with chiefly in conditions which impede the perfect oxygenation of the venous blood, such as the diseases already mentioned. It is also met with in congenital heart disease and mediastinitis, in the former case being accompanied by cyanosis. It is seen occasionally in apparently quite healthy people.

The condition is usually bilateral and symmetrical, but unilateral clubbing of the fingers has been observed in a case of subclavian aneurysm.

Hypertrophic pulmonary osteo-arthropathy, or clubbing of the fingers and toes, with enlargement of the hands, feet, and long bones. The condition is very uncommon, but may occur in association with chronic, usually suppurative pulmonary affections, but not necessarily tuberculous. The enlargement is the result of actual thickening of the bones, due to a toxic periostitis, causing a subperiosteal deposit of bone, which is very striking. It is observed in cases of bronchiectasis, empyema, abscess of the lung and tuberculous cavities.

Rate of Breathing.—Normally there is one complete respiration for every four pulse-beats, or about eighteen per minute. The number of respirations per minute,

and the pulse respiration ratio, vary considerably in disease, according to the conditions present which interfere with the proper oxygenation of the blood and consequently lead to increased stimulation of the respiratory centre in the medulla by the additional carbonic and other acids contained in the blood.

Increased rate of breathing.—The respiration-rate is increased proportionately to the pulse-rate in any febrile condition, according to its severity.

In *acute pneumonia* the respiration-rate increases out of proportion to the pulse-rate, the respiration pulse ratio altering from 1 to 4 of health to 1 to 3 or 2½. This is an important diagnostic sign of acute pneumonia. When the case is taking a favourable turn, the respirations, pulse, and temperature all return to normal simultaneously, and it is always a ***danger-signal*** if there is any departure from this uniformity of improvement. Thus it is a bad sign if pulse and temperature fall to normal and the respirations remain quick: or if pulse and respirations fall to normal and the temperature keeps high, and so on.

In *acute bronchitis* of the larger tubes there is not sufficient obstruction to the entry of air into the lung alveoli to quicken respiration, but when the bronchioles are swollen the mucous membrane narrows their lumen, and the respiration becomes quickened out of proportion to the increased rate of the pulse. In a child of nine we have counted 50 respirations to 120 beats of the pulse when there was some bronchitis of the asthmatic type, and wheezing over the bases of the lungs, but no pneumonia or broncho-pneumonia.

In pleuritic effusion, œdema of the lung, pneumothorax, and abdominal disease pushing up and restricting

the movement of the diaphragm and affecting the bases of the lungs, the respirations are quickened.

In *heart disease* breathing is quickened when the muscle is unable to drive blood sufficiently quickly through the lungs, or in *anæmia* when the blood cannot carry a normal amount of oxygen to the tissues.

Slower rate of breathing.—When the entry of air into the alveolar tissue is difficult—*e.g.*, emphysema, asthma—and the muscles of forced inspiration are working, the duration of each full respiratory movement is increased considerably. The actual number of respirations per minute is less than normal.

Cheyne-Stokes breathing, in which there is a regularly occurring abnormal respiratory cycle, is not met with in pulmonary disease, but generally in some intracranial lesion affecting the respiratory centre. The cycle is a period of no breathing at all, then of respiration beginning in a very shallow way, and gradually increasing in depth until sometimes it is distressing to the patient in its intensity. Respiration then passes off in reverse gradations, until all movement ceases altogether for a few seconds, to begin again and continue as before. The whole cycle occupies about a minute or a minute and a half.

The nature of the breathing gives important information as to the ease or difficulty in oxygenating the blood, which is the main object of respiration.

When the process of oxygenation of the blood and the respiration is normal, the breathing is carried on by easy movements of the intercostal muscles, the ribs and sternum move up and down under their action, and the contractions and relaxations of the diaphragm protrude and relax the abdomen.

Eupnœa is the term given to normal easy breathing.

Dyspnœa.—When there is difficulty in oxygenating the blood, respiratory movements are increased in frequency or more extensive in range, and there is breathlessness or, as the patient may say, the breath is "tight" or "short." Dyspnœa is the term given, in disease, when there is such difficulty in breathing. The painful, catchy breathing of acute pleurisy is not properly termed dyspnœa.

Orthopnœa is the term employed when the patient can no longer breathe when lying down, but has to be in the erect or sitting-up position.

In dyspnœa and orthopnœa the *extrinsic* muscles of respiration come into play.

Dyspnœa may be caused by—

(1) Affections of the alveoli, limiting the amount of the available lung oxygenating surface.

(2) Obstruction to the entry of air into the lung alveoli.

(3) Restriction of the respiratory movements, and consequently of the intake and output of air.

(4) Deficient circulation of the blood, from primary circulatory defects.

(5) Deficient hæmoglobin in the red blood-corpuscles to carry oxygen to the tissues in anæmia.

(6) Abdominal disease leading to an increased content of the abdominal cavity. This pushes up the diaphragm and restricts its movements.

Pulmonary disease causes dyspnœa when there is any reduction in the amount of alveolar tissue available for the circulation of the blood. Consolidation of the lung in acute pneumonia, consolidation and destruction of the lung in tuberculosis, abscess, pressure on the lung from

serous fluid, or pus in a pleural cavity, new growth or aneurysm pressing on the lung, are such causes.

Obstructive dyspnœa from interference with the entry of air into the alveoli. The obstruction may be due to:

(1) *Obstruction of the glottis* from any cause.

(2) *Pressure on the trachea* or *bronchi* from aneurysm or new growths.

> ***Stridor*** is the term given to the noisy respiration produced by these two causes.

(3) *Inflammatory swelling* of the mucous membrane.

(4) *Much secretion* in the bronchi in bronchitis and

(5) *Spasm* of the involuntary muscular fibres of the bronchi and bronchioles in asthma.

Catarrh and swelling of the mucous membrane of the bronchial tubes, especially the medium and small ones, reduce the lumen of the air passages, just as catarrh of the nasal passages in a cold in the head does. Everyone knows how it is impossible to breathe through the nose with a cold in the head. In the same way there is obstruction to the passage of air through the bronchial tubes, which are reduced in lumen by swelling of the mucous membrane.

Dyspnœa from restriction of the respiratory movements.—In *emphysema,* in which the lung is permanently overdistended and inelastic, the chest is permanently in a state of full intercostal inspiration, with the sternum as far from the posterior chest wall as it is at the end of normal inspiration. The diaphragm is also pushed abnormally far down for a similar reason. Further inspiratory movements can only be made by the action of the extrinsic muscles of respiration, especially the sterno-mastoids.

Paralysis of the intercostal muscles and of the

diaphragm from nervous causes also cause dyspnœa, which may lead to asphyxia.

Inability to cough.—When the diaphragm is paralyzed there is none of the normal protrusion of the abdomen with inspiration, and the patient cannot cough.

Dyspnœa from heart disease does not concern us here, but it must be remembered that this may cause and coincide with pulmonary disease.

Thrombosis of the pulmonary artery, which is met with in septic conditions, chiefly after child-birth, generally causes death from extreme breathlessness very soon.

Embolism, or blocking of one of the pulmonary artery branches by a plug of vegetation or blood-clot from the right side of the heart, gives rise to symptoms of acute pleurisy and pneumonia of sudden onset (infarct).

Dyspnœa from a deficient supply of hæmoglobin and red corpuscles (anæmia) does not concern us here.

Abdominal disease, leading to increased content of the abdominal cavity, pushing up and restricting the contraction of the diaphragm, throws the muscles of forced respiration into play.

Expiratory dyspnœa is the term given to the condition when expiration is abnormally prolonged or noisy. This may arise—

(1) In *emphysema* when the lung tissue has lost its normal elasticity through chronic distension, and is unable to expand with inspiration or contract with expiration.

(2) In *asthma* when the air passages are abnormally narrow.

(3) In *obstruction* to, or pressure on, the large air passages, as in laryngeal obstruction, and the pressure of new growths or aneurysm on them.

Orthopnœa.—The erect or orthopnœa position is not so common, generally speaking, in pulmonary disease as in cardiac disease. It is usually adopted by patients with—

(*a*) ***Chronic bronchitis and emphysema,*** so as to allow the freest possible movement of the chronically distended chest by the muscles of forced inspiration and expiration. Here the distended inelastic vesicles keep the chest in a condition of deep inspiration, with the ribs and sternum elevated, and maintain it there. For the ribs and sternum to be elevated farther the muscles of forced inspiration, especially the sterno-mastoids, come into play.

(*b*) ***Asthma,*** arising from whatever cause, also brings the muscles of forced respiration into play in the endeavour to increase the negative pressure in the chest on inspiration, and so force more air through the smaller air passages, which are narrowed by contraction of the involuntary muscle in their walls. The extent to which narrowing of the air passages interferes with the entry of air into the lungs can be understood from the difficulty of breathing through the nose with a cold in the head.

The air hunger in asthma is so great that its subjects will frequently open a window and lean out to get more air.

In extreme cases of orthopnœa the patient grips the arms of the chair, so as to give the muscles attached to the humerus and the ribs a fixed point on which to exert a great effort to elevate the chest.

The **lying-down position** in bed occupied by a patient with pulmonary disease is that which will offer the least obstruction to the movement of the working part of the lungs. In other words, the patient tends to *lie on the*

abnormal side, so as to allow the freest possible movement of the normal lung.

Thus, if a patient lies solidly and constantly on one side, suspect a severe lesion on that side—pleuritic effusion, empyema, lobar pneumonia, or new growths.

He will also occupy a similar position in acute pleurisy, to fix the affected side of the chest, and so restrict painful movements of the abnormal side.

COUGH.

A cough is Nature's method of removing irritating material, generally excessive secretions, blood or serum, and occasionally foreign bodies, from the lower respiratory passages—*i.e.*, those below the epiglottis—and is set up by an afferent stimulus from the seat of irritation. Its mechanism is first the passing of an afferent or sensory stimulus from some part of the body supplied by the vagus to the brain or to the coughing centre in the medulla; this sets up a deep inspiration, and causes closure of the glottis, which is followed by strong spasmodic contractions of the muscles of forced expiration, in attempts to expectorate the irritating material. The abdominal muscles, by contracting forcibly, reducing the cavity of the abdomen, and pulling the lower ribs downwards, play an important part in the coughing mechanism. Patients with a constant cough frequently complain of aching or pain in the abdominal muscles.

A cough is not always of pulmonary origin.—A cough is usually caused by irritation of twigs of the superior laryngeal branch of the vagus in the respiratory passages, especially below the glottis, by some material which it is desired to cough up. It can,

however, and frequently does, arise from stimuli from any part of the body supplied by the vagus nerve, and without there being any pulmonary disease or any irritating material to be removed. Such a cough is generally dry, with no expectoration.

Pulmonary coughing proper occurs when the afferent nerve conveys to the mind the sensation of there being something in the respiratory passages below the glottis which is causing some discomfort. The subject then voluntarily coughs and removes tho offending material. With the removal of the secretion, the sense of irritation ceases, and the impulse to cough passes off for a time.

Reflex coughing, or coughing not of pulmonary origin, occurs when an efferent nerve conveys to the reflex centre in the medulla the sensation of some irritation anywhere in the distribution of the vagus—in the nose, mouth, pharynx, external ear, alimentary canal, bladder, pleuræ, and also from stimulus of cold air on the skin—and a cough with no expectoration follows.

The most common reflex coughs are pharyngeal, ear, or stomach coughs.

Pharynx.—Irritation of the pharyngeal branches of the vagus, especially in the portions behind the pillars of the fauces, from chronic catarrh, may cause a very troublesome, irritable cough. Careful examination by mirror may show a small irritable granulation. If this be removed, the cough caused by it ceases at once. Tobacco is a common cause of a pharyngeal cough. A long uvula is also a likely cause of reflex cough, especially at night, when it falls on irritable regions.

Ear irritation in the external auditory meatus, especially from foreign bodies or dry catarrh, by stimulating the auricular branch of the vagus, will set

up a short, persistent, dry cough. The cough frequently produced by the introduction of a speculum into the external auditory canal is a good illustration of this reflex cough.

Stomach.—Disturbances of digestion may give rise to a hollow reflex cough.

Voluntary and involuntary coughing.—Coughing, pulmonary or reflex, may be a voluntary and controllable act, or involuntary and uncontrollable.

Voluntary coughing.—The simplest form of voluntary coughing occurs when there is hoarseness of the voice because of some abnormal amount of secretion in the larynx. This causes the patient to cough once or twice, or to "clear the throat" by a prolonged, forcible expiration with the glottis nearly closed. It also occurs when there is some catarrh of the trachea or large bronchi, the secretion from which collects in a mass and makes its presence felt by a tickling sensation. A cough removes the foreign material and the source of irritation, but it may have to be repeated soon and frequently, according to the amount of the abnormal secretion.

Involuntary, uncontrollable coughing is often caused when there is a small patch of inflammation in the respiratory passages below the glottis, which irritates in a tickling way. Such a cough is described as a "tickling cough," and is very annoying and even distressing. Very little, if any, sputum is coughed up after a deal of exertion, and the patient may be kept awake most of the night with it.

It also occurs in chronic bronchitis, sometimes in the early morning, and the patient may, as he says, "cough himself blue in the face" with it.

Similar uncontrollable coughing of a persistent and exhausting character may be excited by *local catarrh* of the smallest bronchioles, with no other sign of disease of the lungs. In cases of very early phthisis a slight, moist sound may be heard occasionally at one apex only, and yet the cough may be most distressing. In other cases a few moist sounds may be heard over the bases of the lungs.

In *fibrinous bronchitis*, after a period of dyspnœa, an involuntary paroxysmal cough comes on, and ends in the coughing up of a mass of sputum, which consists of a cast of some of the intralobar air passages. This coughing attack is generally very exhausting to the patient, from its duration and force.

The amount of the expectoration varies in different diseases, and in different stages of the same disease. (See p. 43.)

The time of day in which a cough comes on is important.

In mild cases of tracheal or bronchial catarrh secretion collects overnight, and coughing is more frequent at the time of getting up than during the rest of the day. In healthy people it is quite usual for one or two coughs to be necessary first thing in the morning, to remove secretion which has collected overnight.

When the bronchial mucous membrane is irritable, the colder air of a bedroom will cause a frequent tickling cough, which disturbs the rest very much. This is often so in children, the parents telling you that the child coughs all the night. In these cases there may be some focus of irritation in the pharynx which no cough medicine will remove, some local application being necessary to cure the condition.

With extensive disease the cough persists throughout the whole twenty-four hours, with very little intermission. If some sleep is obtained, the patient wakes to a coughing fit which makes up for lost time.

In other cases of chronic bronchitis some sleep of exhaustion may be obtained in the early morning, with the result that secretion accumulates and a fit of uncontrollable coughing exhausts the patient when he begins to get up.

The duration of a cough is an indication of the acuteness or chronicity of the condition causing it. If it has been present a few days only, we expect to find an illness of recent origin; if it has been present for weeks or months, one of long-standing nature.

Winter cough.—A cough that has been present for several past winters, passing off in the better weather of the summer, must arouse suspicion of pulmonary tuberculosis. The cough of chronic bronchitis and emphysema, though it will improve in the summer, generally persists throughout the year in more or less severe form.

Special forms of cough.—***Brassy cough*** is met with in unilateral paralysis of the laryngeal muscles affecting a vocal cord. This cough occurs most commonly when there is pressure on the left recurrent laryngeal nerve in the thorax, generally from an aneurysm. The voice is rough and the cough hard and "brassy." If there is paralysis of both vocal cords, the cough is hoarse.

Goose or gander cough.—A somewhat similar cough is heard when there is pressure on the trachea or one of the large bronchi from aneurysm of the transverse part of the arch of the thoracic aorta or by new

growth. It may be wheezy as well as brassy, and is very characteristic.

A croupy cough is not uncommon in the laryngeal catarrh of children, which makes the glottis irritable and causes spasm. In this condition it is more alarming than dangerous. It may be associated with large tonsils.

A noisy, barking cough of spasmodic laryngeal origin, hollow and deep in tone, is heard in nervous children, and especially in young females. Attention to it makes it worse.

A similar bark is not uncommon about puberty, especially in boys. It is a short cough at fairly regular intervals during the day, ceasing during the night, and also if the subject's mind is attracted from himself.

Danger of paracentesis abdominis when bronchitis is present.—The part played by the abdominal muscles in coughing must be remembered when paracentesis abdominis for the relief of ascites is contemplated when any bronchitis is present. When there is a considerable amount of free fluid in the peritoneal cavity the abdominal muscles, especially the recti, are stretched, and when they contract in the act of coughing press on the fluid behind them. If the fluid be removed, the stretched abdominal muscles are no longer able to contract sufficiently to exert pressure on the abdominal contents and to pull down the lower ribs, and consequently are no longer able to help the diaphragm and thorax muscles in their expulsive efforts in coughing. The result is that the patient cannot get rid of the bronchial secretion, and may easily be drowned in his own sputum.

EXPECTORATION.

The expectoration or sputum of pulmonary disease varies considerably in amount and character, according to the pathological process causing it.

Quantity.—***No expectoration.***—With dry pleurisy, when the cough may be troublesome, and with reflex coughs, there is no expectoration at all, unless there is some intrapulmonary lesion present also. There may be no expectoration throughout an attack of acute pneumonia or of miliary tuberculosis of the lungs.

Scanty expectoration.—In the early stages of acute bronchitis, in spasmodic asthma, and in whooping-cough, although the cough is very persistent and violent, there is little sputum coughed up.

Free expectoration occurs in chronic bronchitis, broncho-pneumonia, acute pneumonia as a rule, and phthisis with breaking down of the lung tissue, abscess and gangrene of the lung.

Cumulative expectoration is a term which may be applied with descriptive advantage to the expectoration in bronchiectasis or dilatation of the bronchi, fibrinous bronchitis, and some cases of pulmonary abscess and gangrene of the lung. In these cases there is no ordinary frequently recurring cough with a mass of sputum removed with each cough, but a long period without any cough, then a fit of uncontrollably violent coughing, and the expectoration of an ounce or a mouthful or several ounces of sputum. In fibrinous bronchitis the mass expectorated is a fibrinous cast of bronchi and bronchioles. The reason for this collection of secretion may either be that the diseased bronchi become anæsthetic, and it is only when there is enough

secretion to reach a sensitive portion that the cough begins; or it may be a reflex, from distension of the diseased tubes.

Odour.—Sputum is odourless in simple catarrhal bronchitis and acute pneumonia; it has a sickly smell when there is much pus in it, and is offensively foul in those conditions in which it occurs in large amounts, or has lain stagnant in bronchiectatic, tuberculous or gangrenous cavities.

Appearance.—In the early stages of acute bronchitis it is of a mucoid, slimy, sticky, colourless nature, with a more or less number of bubbles in it. It is colourless, thin, watery, and frothy in acute œdema of the lung.

Muco-purulent sputum is seen when there is a small amount of pus present with mucus.

Pus, in more or less amount, occurs in chronic bronchitis and those conditions which give rise to a profuse amount of sputum. The pus, when profuse, has a characteristic sickly yellow colour.

In phthisis the pus may be of a darker greyish colour, and occur in curious discrete nummular masses, which do not coalesce, and which derive their name from their imaginary coin-like shape when seen in water.

Hæmoptysis, or spitting of blood.—Blood is frequently coughed up in pulmonary diseases, especially tuberculosis. It varies in amount and appearance very greatly. Its simplest appearance is when it occurs in streaks in sputum expectorated in the early sticky, dry stages of acute bronchitis, when a good deal of hard coughing may be necessary before any mass is expectorated. This is purely a local injury to a congested mucous membrane or to a dilated venule, and has no

pathological significance, although it may be thought by the inexperienced to be an indication of phthisis.

The other extreme is when a pint or more of bright red, florid, more or less frothy blood is brought up with very little effort, as in true hæmoptysis.

In *acute pneumonia* blood in the sputum varies in appearance from a pale lemon-yellow tint to the pure blood of free hæmoptysis. The lemon-yellow colour is quite distinct from the sickly yellow of pus, and tinges a sticky mucoid material to the characteristic colour. It may also be an orange-yellow, but is most commonly a brick-dust or *rusty colour* in acute pneumonia, again with sticky mucoid material. Almost pure blood may be coughed up in considerable quantity in acute lobar pneumonia or pulmonary infarct; and bright red, frothy sputum in broncho-pneumonia.

Blood often has a *prune juice* or chocolate tint in new growths of the lungs or mediastinum. It may also have this appearance in gangrenous pneumonia.

In aneurysm pressing on the trachea or bronchi or alveolar tissue blood may be coughed up freely, and, if the aneurysm ruptures into one of the air passages, in fatal amount.

Blood may be coughed up in ulceration of the larynx.

Free hæmorrhage from the lungs is most commonly met with in *pulmonary tuberculosis*, but it must be remembered that it is by no means an uncommon occurrence in *heart disease* the result of pulmonary embolism, or when obstruction at the mitral valve orifice and failure of the right ventricle cause congestion of the pulmonary circulation.

Diagnosis.—Hæmoptysis means the expectoration of blood from the respiratory passages below the glottis.

Frequently blood-stained saliva or mucus is brought up, especially first thing in the morning, which arises from the gums or the pharynx, and these sources of blood must be eliminated whenever any patient complains of spitting blood.

Spitting of blood must be distinguished from *vomiting of blood.* Blood coughed up in considerable quantities is often spoken of by the patient as having been vomited, and unless this fact be remembered and guarded against, errors of diagnosis are certain to arise. If the different characteristics of the acts of vomiting and coughing are borne in mind no mistake need be made. The important point is that blood is coughed up over a much longer period of time than it is vomited. The latter may occur two or three times and no more blood be seen, but blood may be coughed up in small amounts after the original large quantity for hours.

There may be free hæmoptysis in chronic *high blood-pressure* of old age.

Blood disease.—Hæmoptysis may also occur in severe blood diseases, purpura, scurvy, pernicious anæmia, and leucocythæmia.

Bleeding from the gums is very common in these diseases.

No disease.—True hæmoptysis may occur when no disease of the lungs develops, and may be due to over-exertion and temporary high blood-pressure, but this diagnosis should not be made without examining the sputum for tubercle bacilli every few weeks when hæmoptysis persists, even if no physical signs of lung disease are to be made out. Spitting of blood may appear on and off in slight amount for months or even a year before any definite sign of tuberculosis appears or bacilli are found.

Fibrinous casts.—In fibrinous bronchitis, which is a very rare condition at Manchester, the large mass coughed up, when floated in water, is seen to be a mucinous and fibrinous cast of smaller bronchi and bronchioles, with pus corpuscles and pulmonary mucous epithelium. It has a branching, tree-like appearance.

In *spasmodic asthma* the sputum is in small round glistening masses, called *perles* by Laennec. When examined in water under the microscope, they are found to contain spirals of mucin, known as Curschmann's spirals, with pus or epithelial cells.

Clear fluid.—In hydatid of the lung clear hydatid fluid, with some daughter cysts, like grape skins, is coughed up.

Bile.—The sputum may be stained with bile when jaundice complicates the pulmonary disease. When a hepatic abscess bursts through the lung, the sputum may be bile-stained or have a curious *anchovy sauce* appearance from an admixture of blood with pus.

IV

PALPATION

Vocal or tactile fremitus.—If a hand of the observer be placed on the front or back of the chest over the lungs when the patient is speaking, the vibrations of the *voice produced in the larynx* are transmitted by the air passages, trachea, and bronchi, and by the alveolar tissue of the lungs to the surface of the chest. The vibration thus felt is spoken of as vocal fremitus, or tactile fremitus.

For the production of this fremitus the voice must have a definite degree of vibration or carrying power, and it must be produced in the larynx, and the air passages must be open. A thin, feeble voice is not transmitted, and in women and children vocal fremitus is very often not to be felt. The sounds "ninety-nine," produced in a deep voice, have good vibratory characters, hence their everyday employment in clinical medicine. If fremitus is not felt with a thin voice, it may be if the patient is asked to say "ninety-nine" in a deep chest voice.

In eliciting the physical sign the hand (which must be warm) must be applied closely to the chest in the desired region, and, as in percussion, identical regions over both lungs must be compared. For a reliable result to be obtained the hand *must not be placed across the middle line* of the body, front or back, and it is even better to keep an inch away from the middle line,

because vibrations from a healthy carrying voice travel well up to the middle line, or over it sometimes, and some fremitus may be felt by part of the hand, and give rise to misleading impressions as to its presence over the suspected area.

Sometimes the whole hand is too much to apply at once, because changes in fremitus may be restricted to a limited area, the rest of the lung covered by the hand being normal. In this way, again, some fremitus may be felt by part of the hand, and mask any absence under the rest of it. It is therefore sometimes necessary to use the palm only. Much adipose tissue over the chest will interfere with the transmission of vocal fremitus.

N.B.—Normally the *vocal fremitus is more marked on the right side* than on the left side.

The reason given for this is that the right bronchus is larger than the left, as it supplies a larger lung. The occupation of so much of the left half of the thorax by the airless heart has probably something to do with it.

Any departure from this condition must be investigated carefully, and its cause ascertained.

For the normal production of vocal fremitus it is necessary (1) for the bronchi and smaller air passages to be open; (2) for the lung tissue to be spongy and (3) in contact with the chest wall; and (4) for the pleuræ to be normal—that is, not thickened—and to have no fluid in their cavities. Changes in these conditions will result in absence or increase in strength of the vibrations.

Diminished or absent vocal fremitus.—(1) *Secretion filling the bronchi and smaller air passages,* as in some

cases of acute pneumonia and œdema of the lung, causes absence of vocal fremitus.

(2) *In compression of the spongy portion of the lung,* and obliteration of the air passages in it, fremitus is absent. The commonest cause of this is fluid in a pleural cavity compressing the lung sufficiently to obliterate the air passages in the compressed portion.

(3) *Displacement of the lung from contact with the chest wall,* by fluid in the pleural cavity in too small amount to compress the lung, or thickening of the pleura, will diminish the physical sign.

In acute pneumonia, when there is a *thick layer of lymph* (quarter of an inch) on the surface of the solid lung, separating it from the chest wall, and the bronchial tubes of the affected lobe are full of exudation, fremitus is absent.

New growth of the pleura invading the lung or obliterating the air passages will also diminish it.

(4) *Air in the pleural cavity,* displacing the lung from the chest wall, also causes absence of fremitus.

Increased vocal fremitus.—Any abnormal condition which facilitates the carrying of the vibrations of the voice from the larynx through the bronchial tubes to the surface will cause an increase in the vocal fremitus.

Consolidation of the lung with open air passages will increase the fremitus. This is very well marked in acute pneumonia, when the alveoli are filled with inflammatory exudation.

If, however, the air passages are *blocked with* much bronchitic *secretion* in acute pneumonia, fremitus may be absent.

In pleuritic effusion, when the spongy tissue of the lung is compressed, *but the air passages are left open,*

vocal fremitus is increased. This leads to difficulties in diagnosis between pneumonia and pleurisy with effusion or empyema, and an exploratory puncture with a needle may be necessary before a diagnosis can be made.

It may be thought by the student that fluid secretion in the bronchial tubes in these cases would be revealed by the presence of rhonchi, râles, or bubbles (*q.v.*); but as no air can enter the lung supplied by them, these adventitious sounds cannot be produced.

Palpable pleural friction.—When the pleural surfaces are roughened and thickened by dry inflammation—dry pleurisy—there is often a rub sufficiently well marked to be felt during the respiratory movements. It conveys the sensation of two leathery surfaces rubbing together.

In simple acute pleurisy with lymph exudation the rub is scarcely ever palpable.

Rhonchial fremitus.—Rhonchi or wheezing, when marked in bronchitis, can sometimes be felt, but the sign is of no diagnostic importance.

Pulsation may be felt when there is an empyema of the left lung, localized by adhesions and in contact with the pericardium. The heart-beats are thus transmitted to the surface in the intercostal spaces. These pulsations are up and down and not expansile in character.

Pulsations from *aneurysm* of the intrathoracic vessels, especially of the aorta, are often felt; they are expansile in character.

V

PERCUSSION

Percussion is a most valuable method of physical examination, by which changes in the amount of air in a lung can be made out with considerable certainty. It depends on the note which is yielded when the surface of the body over any air-containing organ is struck or tapped with the finger or small rubber-tipped hammer, in the same way that a note is obtained by beating a drum with a drumstick. At first a hammer was used to strike on a small rectangle (2 inches by 1) of ivory—or pleximeter—which was pressed against the body in the desired position. This method is now almost entirely replaced by using the middle finger of the right hand as a hammer, and the middle phalanx of one of the fingers of the left hand pressed against the body as the pleximeter. The hammer finger must be held rigidly in a curved position, and the stroke made by free movement of the whole hand from the wrist, not pecks by the finger moving alone or blows from the forearm working from the elbow. The proper stroke can be practised by laying the forearm on a table, and working the hand from the wrist as described above. The stroke should be of a staccato nature—that is, not too quick and light, and not too slow and heavy.

Tympanitic note.—The most drum-like note is obtained by percussing over a single-cavity organ, especially the stomach or the intestines, when, as so

often happens, they contain air or gases. The note thus given is called tympanitic or drum-like. It has the lowest pitch (that is, has the fewest number of vibrations per second) and is the most prolonged of percussion notes.

The term "tympanitic" is not a satisfactory one for describing *lung* percussion sounds, as nothing really like the tympanitic stomach or intestine note is heard in pulmonary percussion. The nearest to it is heard over a pleural cavity, distended with air in pneumothorax, which really is drum-like. A lung generally overdistended with air, in emphysema, does not yield a true tympanitic note like that of the stomach.

Airless note.—The other extreme is obtained by percussion over the airless muscles of the thigh, or over bone, when no drum note whatever, but merely a dull, toneless sound, is heard. This has the highest pitch (that is, has the most vibrations per second) and is the shortest of percussion notes.

Percussion in the normal body *over the heart or liver* also yields a dull note, but as these organs lie in contact with air-containing organs—lungs, intestines, stomach—an absolutely dull, airless note is not always obtained in abnormal conditions.

The other airless organ, the *spleen*, is difficult to get a dull note over, unless it be considerably enlarged, as the normal lung lies between it and the wall of the thorax and the distended stomach is closely opposed to it.

Routine method of percussion.—The most delicate results of percussion are obtained by laying the pleximeter finger in the interspaces parallel to the ribs on the front of the chest and axillary regions, and not on the ribs themselves. On the back of the chest the intercostal spaces, where not covered by the scapulæ,

are often too narrow for a finger to lie between the ribs, and the percussion note must then be obtained over the ribs.

The pleximeter finger must be applied firmly over the part which is to be investigated, and must lie flat in perfect apposition with the chest wall.

Percussion does not require much force for superficial lesions, especially in thin people and children, but firmer, more vigorous strokes are necessary for deeper lesions and in patients with thick body walls.

When comparing the resonance of the two sides of the chest, the pleximeter finger must be *placed over exactly similar situations on the two sides*—that is, in the same intercostal spaces or over the same rib, as the case may be—and short, quick contrasts should be made. The pleximeter finger should not touch the sternum in front, as by means of the sounding-box effect of this bone conditions in the other lung will influence the note.

Sense of resistance.—Whilst the information which is conveyed to the ear by percussion is of great diagnostic value, that conveyed by the sense of "resistance" of the chest to the finger on it and the percussing finger is hardly of less importance. For instance, note the different impressions which are conveyed to the tactile senses by percussing over a distended stomach, and over the thigh or liver, and then over the lung tissue proper.

To appreciate this important method of examination it is necessary to use a light method of percussion, and impressions of elasticity or softness and solidity or hardness, and a halfway condition between these two respectively, are received.

Hæmoptysis.—Any case of phthisis with hæmoptysis (*q.v.*) should be examined by very slight percussion;

indeed, the less such a case is examined the better. Recurrence of hæmoptysis may come on any time, and if it should do so by coincidence after examination by heavy percussion, the observer will certainly be blamed for it. On no account should an attempt be made to elicit a cracked-pot sound (*q.v.*) when hæmoptysis is present.

Small patches of consolidation.—These are sometimes extremely difficult to make out by percussion, especially when there is some compensatory emphysematous distension of the intervening lung tissue. Very often we have to rely solely on the auscultatory signs to identify these conditions or to *x*-ray examination by experts.

Position of the patient for percussion. — Some clinicians prefer to examine the patient in bed; others in the sitting or standing positions. In doubtful cases it may be necessary to try all these. The muscles must be relaxed as much as possible.

For the anterior regions the arms should hang limply by the side if the patient is sitting or standing, or lie loosely by his side if he is in bed. The apex of the lungs above the collar-bone must be examined carefully.

In examining the back, the shoulders and scapulæ should be moved far forward, so as to have as much interscapular space as possible, and the head and body should incline forwards slightly. With the arms folded across the chest and hanging loosely down with lax muscles, and with the shoulders far forward, the intervening muscle is reduced to a minimum of thickness, and the value of the results obtained is increased.

In this way the *apices* of the lung are best investigated from the back.

Another position is for the patient to put one foot on a chair and then incline forward loosely and limply.

The *apices of the lungs* above the clavicle must be carefully examined by anterior and posterior percussion. Anteriorly lay the finger above and parallel to the collar-bone, and posteriorly hold it right over the upper fold of the trapezius muscle on to the supra-clavicular region. Then percuss above the inter-scapular regions away from the scapula. Adopt identical measures for both apices, and take short and quick contrasts.

Slight changes may be made out if the apex is percussed at the end of expiration when the breath should be held for a few seconds.

N.B.—The right apex above and below the collar-bone region is less resonant than the left apex, and any alteration in this comparative condition in a patient must excite suspicions of disease in one or other apex. All the other areas of the lungs should be carefully percussed.

Axillary regions.—To investigate the very important axillary regions—on the right side the apex of the middle lobe of the lung is here—the arms should be elevated and placed resting over the vertex of the head.

N.B.—The *apices* of the various lobes of the lungs are common seats of *tuberculosis*.

Characteristics of a percussion sound.—In describing a percussion sound we do not speak of its *loudness*, for this depends entirely on the force of the percussion stroke used to produce it, but we do refer to its duration, pitch, and tone.

Duration.—The note obtained over an abnormally distended lung lasts longer than that from over a normal lung, and longer still than the heart or liver note.

Pitch refers to the number of vibrations of the note,

the longer notes having fewer than the shorter notes. Thus the overdistended lung is of the lowest pitch, and the consolidated lung, the liver, or the heart, of the highest, with the normal lung note and its slighter modifications between.

Tone.—Resonant and dull are the two most convenient contrasting terms of the tone of percussion notes: a normal lung being resonant, an overdistended lung being hyper-resonant, and a consolidated lung or the heart or liver being dull, with intermediate variations as before.

Normal lung note.—Percussion over the normal lungs, well away from the heart and liver, gives a characteristic note obtained nowhere else, which is neither a tympanitic nor a dull, toneless note. This is only to be expected when it is remembered that the lungs consist of innumerable little air-containing vesicles surrounded by the interstitial lung tissue. It comes between the tympanitic note and the dull, airless organ note in pitch (or number of vibrations per second) and duration.

The normal lung note cannot be described in words; its characteristics must be learnt by experience. It can be heard best by percussion over the subclavicular region in the second interspace; and over the bases of the lungs when the chest wall is not too muscular or adipose.

In the *left lung* anteriorly the heart begins to influence the note about the third rib in the parasternal line, and when the left edge of the heart is in relation to the lung, about the mid-clavicular line. It is clear over the whole of the posterior region down to about the ninth interspace. In the lower part of the posterior axillary region from about the seventh rib the spleen

influences it; and in the middle and anterior axillary regions the true tympanitic stomach note is generally heard on percussion there.

In the *right lung* anteriorly the liver influence comes in about the sixth rib, and posteriorly about the ninth interspace.

The terms *superficial* and *deep dulness* are applied to the notes obtained by percussion over a solid organ with and without lung intervening respectively, and refer to the investigation of the solid organ, not of the lung.

Variations in health.—The normal percussion note is affected considerably by the condition of the bones and the soft tissues of the chest wall.

The *bones* of the chest wall and the scapulæ affect the note, as can be seen by percussing over a lung in the dead subject when in the chest and when removed from it.

A percussion note is more easily obtained in children before the bones have attained their full adult development; and in normal young children "with yielding bones" even a sign of serious disease in the adult, the cracked-pot sound, may be elicited.

The *scapulæ* deaden the note very considerably.

Percussion *over the sternum* is never satisfactory, because the bone is attached to both sets of ribs, and acts as a sounding-box for the whole anterior aspect of the chest.

The amount of muscle and adipose tissue over the ribs also influences the note; the more of them the less easily is it obtained.

Abnormal percussion notes.—Since the normal lung percussion note depends on there being a normal

amount of air in the alveoli and air passages, and a normal amount of interalveolar and interstitial tissue in the lung covered by normal pleuræ, any condition affecting the normal amount of air in the lung will affect the normal percussion note.

1. ***More air than normal in the alveoli,*** when the percussion note becomes hyper-resonant, or increased in resonance, varying, of course, with the amount of additional air space in the lung. This change in the amount of air may occur in both lungs in *emphysema:* in one lung to compensate when the other is airless and out of action or in part of one lung adjoining an airless portion of the same lung.

The hyper-resonant note is heard in its most extreme form in *pneumothorax,* when it may be a real stomach tympanitic note.

2. ***Less air than normal in the alveoli,*** when the note becomes less resonant, whilst still retaining some air note. The common conditions in which this occurs are—

(*a*) *Congestion of the lungs,* when there is more blood than usual in the interalveolar tissue, and secretion or serum in the bronchioles, especially in the lower parts of the lungs, and therefore less room for air in the vesicles.

(*b*) *Thickened or adherent pleuræ,* which often occur together. These conditions not only diminish, if only slightly, the amount of air in the lung, but they also interfere with the production of the normal percussion vibrations of the lung beneath.

(*c*) *Chronic interstitial pneumonia,* or pulmonary fibrosis, or fibroid phthisis, in which the alveolar tissue is gradually replaced by fibrous interstitial tissue. The

amount of resonance will vary with the extent of the fibrous invasion, until eventually there is none at all.

(*d*) *Broncho-pneumonia.*—Impaired resonance is obtained in broncho-pneumonia, where we get lobular consolidation with air-containing lung around.

3. ***No air in the alveoli,*** when there is no resonance and the note is dull. The common diseases in which this occurs are—

(*a*) *Consolidation diseases,* croupous pneumonia, tuberculosis, new growth; or

(*b*) *Compression by fluid* in the pleural cavities.

In these conditions there is no air in the alveoli involved, and the percussion note over the abnormal areas is the dull, toneless, solid one of an airless organ. When the pleuritic fluid is limited to the lower half of the pleural cavity, some air note comes into the tone over the thin upper layer of fluid.

In chronic interstitial pneumonia or fibroid phthisis the percussion note may be without any resonance, or may be dull.

The dull note does not vary in extent by changing the position of the patient in any of the above conditions.

When the upper part of a lung is not solid or compressed, but the lower lobe is, percussion under the collar-bone yields the skodaic resonance.

Skodaic resonance, subtympanitic resonance.—This peculiar note is heard over local emphysema or air distension of a lung contiguous with an airless portion of the same lung. Its characteristics cannot be described, but they can be imitated approximately by percussing over one's own cheek with the mouth open, and the amount of air in the mouth varied by moving

the lower jaw up and down and by distending or flattening the cheeks.

It is called skodaic resonance, after the clinician, Skoda, who first described it, and also subtympanitic resonance.

Skodaic resonance is heard typically over the upper lobe of a lung in the subclavicular region when the lower lobe is compressed by a large pleural effusion, or when the upper lobe of the lung is becoming consolidated from within outwards or from behind forwards, and there is still some of the superficial portion containing an unusual amount of air.

Cracked-pot sound.—A peculiar note, somewhat similar to that obtained by clasping your hands at right angles to each other and making an air-filled hollow space between the fingers and palms, and then striking this against your knee, is to be obtained when percussing over a superficial ***cavity*** in the lung in an *infraclavicular region* during expiration, the patient's mouth remaining open. The forcible driving out of air from the cavity in this manner makes a peculiar "squishing" noise. It requires considerable strength of blow to obtain this cracked-pot sound, and, as it has been stated above, it must not be tried for without caution, and not at all if there is any hæmoptysis, for a fatal hæmorrhage may occur after its demonstration.

It may be heard when percussing over isolated patches of air-containing lung or bronchi amidst consolidated lung.

It may be obtained *normally* in crying babies and young children with elastic chest walls.

Wintrich's sign.—The pitch of the note obtained by percussion over a cavity is higher when the patient's

mouth is open than when it is closed. This is known as Wintrich's sign.

Change of position of dull note.—In pleural effusion, unless in addition to fluid there is free air in the pleural cavity, the area of dulness does not vary with the position of the patient. Free air in the pleural cavity always tends to pass to the highest point, so that if a patient with air and fluid in the pleural cavity (hydro-pneumothorax) gets on to his hands and knees, and then bends the arms until the face and breast rest on the bed, the dulness due to the fluid will be found to have moved from the base towards the apex of the lung.

In *moderate pleural effusions*, when the patient is sitting up, the upper limit of dulness is lower over the back of the chest than it is in the axilla or at the front of the chest.

Paravertebral dulness or Grocco's sign.—With large unilateral pleural effusions it almost constantly happens that there is a triangular area of dulness close up to the middle line of the body at the back, and on the side *opposite* to the effusion. It is sometimes difficult to make out clearly. It is known as Grocco's sign, and has been attributed to muting of the vibrations of the vertebral column by the pressure of the fluid, and to pushing over the mediastinum to the unaffected side.

VI

AUSCULTATION

Normal Breath Sounds.

The sounds produced by respiration in health are of two broad types, bronchial and vesicular.

Bronchial breathing is most typically and easily heard over the larynx and trachea, and over the large bronchi where they are nearest to the surface—that is, over the manubrium sterni in front and at the level of the fourth dorsal spine in the interscapular spaces behind.

Causation.—It is produced by air passing through the glottis, and heard over the larynx, trachea, and large bronchi, and can be imitated by putting the vocal organs into position for phonating the "ch" of the Scotch word "loch" or the syllable "ker," and breathing in and out. With it both inspiration and expiration are equal, and equally audible, and there is a pause between its first and second phases.

Tracheal breathing is an exaggerated form of bronchial breathing, and is heard, as its name indicates, over the trachea. The student can easily familiarize himself with its character by the use of a binaural stethoscope on himself.

Vesicular breathing is best heard over the bases of the lungs, but is nothing like as distinct as bronchial breathing, and is rather difficult for the student to hear at first. It is of a soft, breezy, sighing character, and

the sound of inspiration is about three times as long as that of expiration, with no interval between its phases. Inspiration increases in its audibility towards its termination, and expiration fades away rapidly from its maximum intensity at its beginning.

Inspiration and expiration are made more audible when a deep breath is taken.

The ***causation*** of vesicular breathing is not very clear, but very probably it is explained by the passage of fine columns of air from thousands of minute bronchial air passages into comparatively larger masses of air in the terminal alveolar spaces—like blowing up so many minute footballs. This would explain the increase in audibility as the alveoli become distended.

If this is so, it is difficult to understand why only part of the expiratory phase of respiration—which is equal in length to or longer than inspiration, as is proved by listening over the trachea—is heard. It may be that a respiratory sound produced in the way this is, is, like the heart murmurs, carried best in the direction of the current of air causing it. Thus inspiration will be well heard, whilst the current of air of expiration, passing away from the ear as it does, becomes inaudible before the movement has ceased.

Another explanation of the causation of vesicular breathing is that it is the glottis sound conducted to the surface through the spongy tissue, and diminished in intensity by being obscured by the feather-bed character of the spongy tissue.

Here, again, it is difficult to understand, if this is so, why vesicular expiration, which is of equal duration and intensity to glottis inspiration, is only a third or fourth the length of vesicular inspiration, and why there is no pause between the two sounds.

There is no pause between inspiration and expiration over the spongy lung tissue, because the natural elasticity of the alveolar tissue of the lungs forces air back from the alveolar spaces as soon as distension by inspiration ceases.

The pause between inspiration and expiration of bronchial breathing is probably explained by the momentum of the inspiratory mechanism continuing the distension of the spongy part of the lung after movement of air through the glottis ceases or becomes so feeble as to produce no sound. Then an equally short pause ensues before the expired air moves with sufficient force through the glottis to cause a sound.

Puerile breathing.—In children (*puer*=a boy, hence the name) the normal vesicular respiratory murmur is much louder than in adults, and expiration is also more marked and prolonged. This is due to the great elasticity of the lungs, the thinness of the chest walls, and the closeness of the lungs to the ear. Loud vesicular breathing in adults is called puerile because of its resemblance to the normal condition in childhood.

Variations in vesicular breathing.—Inspiration, especially in stout people, is sometimes very indistinct in quiet breathing, and *expiration may be inaudible in health*. If inspiration be diminished or inaudible on forced breathing, it is generally a sign of disease, whilst inaudible expiration is not necessarily a sign of disease.

Jerky or cog-wheel breathing.—Normally inspiration is uniformly continuous, but with nervous people it may take place in a series of little jerks—jerky or cog-wheel breathing. When this is heard generally over both lungs, it does not mean any disease as a rule. If it is heard over one part of one lung only, especially the apex, it

almost certainly means that the lung tissue has lost its elasticity and has become fibrosed or invaded by tuberculous masses, or that the alveoli walls stick together and require abnormal respiratory force to separate them.

Breath Sounds met with only in Disease.

Prolonged expiration in adults is only met with in disease, and most commonly in emphysema, where the lungs and the thorax are in a constant state of full inspiration, and very little air can enter the alveoli with each inspiration. The vesicular murmur is *less marked* in its inspiratory phase and *actually* or *relatively prolonged* in its expiratory phase.

Prolonged expiration is also a marked feature of asthma. Breathing in these cases is very noisy, and can be heard when standing at the bedside and without any stethoscope. Heard with the stethoscope, inspiration and expiration are very loud, and expiration is unduly prolonged. There is no bronchial character about it.

Diminished vesicular breathing.—When the alveoli are compressed by fluid in the pleural cavity or by new growths, or filled with inflammatory material, little or no air can enter, and the vesicular murmur is accordingly diminished or completely lost. It is also diminished when the pleuræ are thickened and adherent.

Tubular breathing is the term given to the modification of bronchial breathing heard over consolidated lung with free bronchial passages, and most typically in *acute pneumonia.*

Some writers call it bronchial, but it has none of the "ch" characters of bronchial breathing. It is softer and more blowing, and can be imitated by breathing in and out through the mouth, and making a soft "coo"

sound with none of the hard bronchial "ch" or "ker" characteristics. It is heard in consolidation of tuberculosis, when the bronchi are not obliterated, and when the lung tissue is compressed by fluid in the pleural cavity sufficiently to make it airless, but not sufficiently to close the bronchial passages in the compressed area.

N.B.—It is always accompanied by whispering pectoriloquy and by increased vocal fremitus.

Indeterminate or ***indefinite breathing*** is the term given by some writers to a breath sound like bronchial breathing, but without its "ch" or "ker" harshness, and not as markedly hollow as tubular breathing; by others to any breath sound which does not agree with the bronchial, tubular, or vesicular or puerile types. We rarely find occasion to want to use such a term unless it be in cases where there is noisy breathing which arises from pressure on the air passages.

Amphoric breathing is a peculiar, somewhat musical form of breath sound, which is heard when a comparatively thin column of air enters a large collection of air in a ***cavity.*** It is heard most typically in pneumothorax, when there is a connection between the lung and the air in the pleural cavity. A good imitation of amphoric breathing is heard when an air-cushion is being blown up, and especially when it is nearly distended, and there is some pressure on the contained air from the walls of the cushion, or when a thin column of air is blown into the mouth of a vase (amphora) with a mouth which is narrow compared to the body of the vase. *A modification* of the pneumothorax amphoric breathing will be heard when a *small bronchus opens into a large cavity* connected with the surface of the lung by consolidated tissue.

Metallic tinkling is a clear ringing sound of metallic quality, which is produced when a sticky bubble bursts or a drop of fluid falls into more fluid in a large cavity containing air and fluid, pus, serum, or blood. It is heard in hydro-pneumothorax and over a large pulmonary cavity. It may also be heard after the succussion or splashing sound is produced. It is audible during ordinary breathing, but is often better elicited by deep breathing, loud speaking, or coughing. The sound has been likened to that produced by drops of water falling from a height upon the surface of a little water contained in a glass vessel.

Coin sound is heard in a similar condition when the stethoscope is placed over the chest and whilst listening with it tapping a large coin laid flat on the chest near the stethoscope with another coin.

VOCAL RESONANCE.

The spoken voice sounds produced in the larynx as heard with the stethoscope over the lungs vary as much in health as the glottis breathing does, according to the part investigated. They are very loud over the trachea, where the sounds made can be distinguished, less so over the bronchi, and so much damped down by the feather-bed or spongy character of the alveolar parts of the lungs as to merely convey an impression of vibration to the ear, similar to that felt by the hand as vocal fremitus.

Bronchophony.—When there is a patch of consolidation around a bronchus which is not filled with secretion, the voice sounds are audible in an altered form, in that they are concentrated and their pitch elevated, both changes together conveying the impression that

the sounds are produced at the end of the stethoscope. To this sound the term bronchophony is given, and it implies the conduction of the sound of the voice rather than the actual spoken words.

The auscultation of the laryngeal voice sounds is not of much assistance in diagnosis of pulmonary disease, because from their vibratory nature they carry too widely in health for delimiting changes of disease; but the conduction of articulate speech, especially of whispered sounds, is of very considerable value.

In a normal condition, *words whispered* in a very quiet way *by the lips* are quite inaudible over any part of the spongy tissue of the lung, but with consolidation of this tissue and patent bronchi a remarkable change takes place.

Whispering pectoriloquy is the term given to the physical sign obtained when the conditions in the chest make it abnormally easy for a sound produced at the lips to be carried to the surface of the chest and the ear of the observer through the lung. For its production ***some lung consolidation, with patent bronchial passages passing through it,*** is necessary, and as these are the ***same conditions necessary to hear tubular breathing,*** it follows that whispering pectoriloquy and tubular breathing can always be heard over the same spot. The way to secure the sign is first to find a patch of tubular breathing, and then to ask the patient, with his head turned away from you, to *whisper with his lips* very gently—"one, two, three." When this is done the sound is very clearly heard, as if *whispered right into the mouth of the stethoscope,* and not where it is produced, 12 inches away.

It is sometimes a useful aid to finding indistinct

tubular breathing to listen over a suspected spot and ask for the whisper.

There is no mistaking the whispered sound coming through the air to the free ear of the observer for the physical sign.

It is heard best in the consolidation of *acute pneumonia* with the air passages in the consolidated area open and *tuberculous cavitation with consolidation* about it. It is also heard in *fluid in the pleural cavity* compressing the lung tissue, but *with the bronchial passages open.*

Ægophony is the term given to the modified voice sound which is heard when there is *serous fluid in a pleural cavity,* extending about halfway towards the apex. In such a condition, if the stethoscope is placed over the upper limit of the effusion, and the patient says "ninety-nine" in an ordinary tone, the voice has a bleating or nasal character as heard through the stethoscope. It seems probable that the thin layer of fluid through which the voice vibrations pass affects it in this peculiar way.

Succussion or splashing sound is heard when there is both air and fluid in a pleural cavity. To obtain this physical sign the patient should be sitting, and told to relax the muscles. A towel is laid over the bare skin, and the ear of the observer placed directly on the towel and firmly applied to it. With his arms about the patient the observer then "shakes" the trunk of the patient, or moves it vigorously about, his ear being all the time kept closely applied to the chest. In this way the fluid and air in the chest can be made to splash, and produce an easily audible splashing sound.

Post-tussive suction is a sucking noise resembling that produced by an india-rubber ball which is opening

again after being compressed. It is a very important sign, in that its presence denotes pulmonary cavitation. With the stethoscope on the chest over the cavity it is heard immediately after a cough.

ADVENTITIOUS SOUNDS.

For the production of the normal respiratory sounds, bronchial and vesicular, it is essential that there be no increase in the normal amount of secretion of the mucous membrane of the air passages, no abnormal fluids in the lungs, and no disease of the pleural membrane. When abnormal secretion or blood is present in the air passages, trachea, bronchi, bronchioles, and alveolar tissue, or on the pleural membranes, ***abnormal*** or ***adventitious*** respiratory sounds are heard. This abnormal secretion may be caused by acute or chronic inflammatory processes, œdema of the lungs, or passive venous congestion, and blood may appear in consequence of the erosion or rupture of bloodvessels in the air passages or diseased lung tissue or effusion of blood in infarcts.

Adventitious respiratory sounds.—For all practical and diagnostic purposes we may say that there are four broad types of abnormal or adventitious respiratory sounds, which are caused by the presence of abnormal material in or on the lungs. These, in our opinion, are best called by the following names:

(*A*) ***Moist sounds.***

(1) Rhonchus or wheeze.	Tracheal or bronchial in origin.
(2) Râle or bubble.	Tracheal or bronchial in origin.

(*B*) ***Dry sounds.***

(3) Crepitation. Alveolar in origin.
(4) Friction or rub. Pleuritic in origin.

1. **Rhonchus or wheeze** is a sound which is produced by local obstruction *in the air passages* to the passage of air to and from the alveolar tissue. It is met with generally in *bronchitis,* and is produced when there is only a moderate amount of increased secretion from the mucous membrane, generally somewhat viscid or sticky, which clings to the surface of the swollen mucous membrane; from this it projects, and slightly narrows the lumen and obstructs the free passage of air through the trachea and the large and small bronchial tubes. The projections from the surface are thrown into vibration, like a reed in a wind instrument, or cause the passing column of air to vibrate, producing a uniform sound during the phases of inspiration and expiration.

A similar condition arises when anyone has a cold in the nose. The mucous membrane is swollen, secreting more mucus, and the familiar noises produced by breathing through the nose in this condition are heard. They are practically rhonchi.

The amount of secretion which causes rhonchi is *limited* as compared with that which causes râles or bubbles, through which the passing air bubbles.

Rhonchi can easily be heard by the patient himself, and by the observer standing by the bedside without a stethoscope.

A high-pitched rhonchus may be heard at the other end of a long ward in which the patient is.

Rhonchi can be *felt* under similar conditions through the chest wall by the hand applied as for feeling vocal fremitus—the physical sign known as rhonchial fremitus (*q.v.*).

Rhonchi are of two broad types, both of which suggest undue moistness in the air passages :

(1) ***Sonorous,*** or low-pitched and snoring, produced in the larger bronchi.

(2) ***Sibilant,*** or high-pitched and wheezing, produced in the smaller tubes.

Their *intensity* or *loudness* depends a good deal on the force of inspiration and expiration.

A cough will remove the sonorous rhonchi of the local bronchitis of the larger bronchial tubes, but it has no effect on the sibilant rhonchi produced in the more general bronchitis of the smaller passages.

When the bronchitis which causes a rhonchus is limited in degree, and confined to the larger bronchi, the sound may be only heard over these large bronchi—that is, in the interscapular region about the level of the fourth dorsal spine, and anteriorly on either side of the manubrium sterni. When it is extensive and affects the smaller tubes, rhonchi are heard in profusion all over the lungs, right down to the bases. When there is extensive bronchitis, vesicular expiration is prolonged and more audible.

Rhonchi are heard in *asthma,* and are then said to be produced by narrowing of the air passages through contraction of the involuntary muscle in their walls, but are more likely to be produced by the exudation or catarrh, small in amount though it be, which invariably accompanies asthma, just as they are in ordinary bronchitis.

2. **Râle or bubble** is the term best given to the sound heard when the secretion is more profuse in amount than that which causes rhonchi, and so obstructs *the larger and smaller bronchi* that air has to *bubble* through it.

The fluid in the air passages may be inflammatory secretion, blood, or serum. The bursting of the bubbles causes the sound. The bubbles generally distinctly suggest moisture or fluid in the lungs.

The term râle, or "rattle," was first used to describe the loud, coarse bubbling or rattling which takes place when death is at hand, and a large amount of secretion collects in the trachea. The patient is too feeble to cough the secretion up, and the air bubbles in and out through it, producing the so-called death rattle. Such a condition is not uncommonly heard for a few hours before death in the last stage of a fatal attack of acute pneumonia, and in any other condition in which secretion collects in quantities and cannot be expectorated. The secretion in these cases becomes so large in amount that the patient becomes cyanosed, and death is partly caused by his being drowned in his own sputum.

Râles or bubbles are met with when the bronchitis is catarrhal in nature and produces free secretion; when there is blood in the lungs, or effused serum from acute pulmonary œdema.

Râles or bubbles do not produce a continuous unbroken sound like a rhonchus, but, as the name suggests, an interrupted bubbling one, lasting throughout the phases of inspiration and expiration. They generally suggest moisture or fluid in the air passages, but those heard in pulmonary tuberculosis may sometimes be drier and hard in character.

Râles or bubbles vary in character according to the conditions under which they are produced, and of the condition of the adjacent or surrounding lung tissue.

They are heard at their *loudest* when produced in a bronchus near the surface of the lung, or when there is some consolidation favouring conduction of sound to the

surface between the bronchus and the surface of the lung, as in tuberculosis. Whatever their character, they are heard better with forced breathing than with quiet breathing.

They are *large* when produced in one of the large bronchi or the trachea, and *small* when produced in the smaller bronchi.

They are *low* in pitch when the secretion is thin, and *high* in pitch or more crackling when it is viscid and sticky.

One form, called the ***consonating râle*** or ***bubble,*** is produced in sticky secretion in a bronchus or cavity with consolidated lung between it and the surface of the lung, which favours its conduction to the stethoscope. This is the highest in pitch and hardest in sound of all. It is this râle which may simulate a dry, crackling crepitation or even—to some students—a pleuritic rub or friction. With it tubular breathing and whispering pectoriloquy will generally be heard.

Rhonchi and râles often occur *in the same patient,* especially when the bronchitis is general, involving the large and small tubes, and producing a good deal of secretion in the smaller tubes.

A *cough* and the expectoration of a mass of sputum *alters the character* of the sounds heard, but does not clear up the râles or rhonchi completely. This is an important aid to their recognition.

3. **Crepitation.**—This term, in our opinion, should be confined to the adventitious sounds arising *in the alveoli* only, not in the air tubes, and produced by the separation during inspiration of the walls of the alveoli, which stick together when viscid secretion lines them, or by the movements of alveoli with changed walls.

Crepitations are fine and coarse in character.

The fine crepitation is heard during inspiration, and in its most typical form in the invasion and again in the resolution stage of acute pneumonia. It is like a rapidly produced minute crackle, and can be imitated by rubbing a bunch of your hair between the fingers close to your ear. This very fine crepitation is called the *redux crepitation* when heard in the stage of resolution of pneumonia. The mechanism of its production can be illustrated very clearly by moistening the tips of the thumb and index fingers, pressing them together firmly, and then, holding them close to the ear, rapidly separating them. A more or less sticky, moist click is thus heard. The character of this sound can be varied by the amount of the surfaces of the finger and thumb which are pressed together. Substitute in your mind for finger and thumb the surfaces of thousands of microscopic alveoli thus forced apart from sticky cohesion, and the mechanism of production of the fine, hair-rubbing redux crepitation is understood.

This fine crepitation can be heard in any conditions in which the alveolar walls cohere. Acute pneumonia is the commonest condition; it may be heard in œdema of the lung, and in lung which has been compressed or collapsed, but into which air can enter on forced inspiration.

One or two crepitations may be heard over an apex of a lung in which there is *tuberculosis*, especially on deep inspiration after a cough.

In normal lungs it can be heard in any patient in bed who has been lying on his back for some time and breathing quietly with shallow tidal-wave respiration. If such a patient be asked to sit up, and the stethoscope be applied over the base of the lungs, and a deep inspiration be taken, this fine hair-rubbing

crepitation will almost certainly be heard, for the one breath, if not for subsequent ones. Here probably we have the very mildest form of hypostatic œdema and coherent air vesicles, which one good inspiration clears away.

Coarse crepitation can be heard over the thin edges of emphysematous lungs, which have lost their elasticity, and in which the alveoli are abnormally large and their walls fibrous or inelastic. They can also be heard over portions of the posterior surfaces of the lungs of elderly people, sixty or more years of age, who have had recurrent attacks of bronchitis. They are there when there is no bronchitis at all, and I have watched them clinically in some patients for several years.

This coarse crepitation is heard in both phases of respiration, and is hard, dry, and crackly, suggesting some stiffening change in the character of the alveolar walls, and not the presence of moisture.

When these inelastic distended alveoli occur in the edges of the lungs and overlie the heart, the movements of the heart produce a similar crackling sound in them.

Similarity between râles and coarse crepitations. —It is sometimes difficult to distinguish between râles, especially consonating râles, and coarse crepitations, especially when both occur together in the same case, but the difficulty is minimized by taking into account the other clinical symptoms and physical signs which are present.

If there is no expectoration and the sounds are heard over the edges of emphysematous lungs, or over the bases in elderly people, the sounds are most probably crepitations.

If there is expectoration, and there is some consolida-

tion which facilitates the conduction of sounds produced within it to the surface, the sounds are most probably consonating râles. They are hard and crackling, and it is this form of râle which so simulates a coarse crepitation.

Some students mistake the hard consonating râle or coarse crepitation for a hard, dry, pleuritic rub, but the latter is more of a to-and-fro sound with peculiar characteristics.

4. **Friction sound.**—This sound is produced *on the surface of the lung.* In health the apposed costal and pulmonary pleural surfaces are sufficiently lubricated to glide over each other noiselessly during respiratory movements or coughing. When, however, the pleural surfaces become inflamed and roughened by inflammatory exudation, and there is lymph exuded, instead of moving noiselessly over each other they rub and produce a sound of *friction*, or a *rub* which is audible on auscultation. It is also accompanied by severe pain, or a stitch in the side on breathing or coughing. In slight cases it may require a deep breath to bring the pain out, and this the patient is often unwilling to take ; indeed, he often restricts as far as possible his respiratory or coughing movements, and takes short, shallow breaths.

Varieties.—Pleuritic friction varies in character from a fine sound very like the fine crepitation to a coarse, leathery, dry rub, which is felt by the patient and is palpable by the observer. The rub may occur with both inspiration and expiration, but is usually most distinct at the end of deep inspiration. It is superficial, *and is not altered by a cough,* like râles are or alveolar crepitation may be.

Pleuritic friction may be heard over any part of the chest, wherever the pleurisy occurs, but is generally

most distinct over the lower portion of the lungs. It may be limited in area of audibility to a small patch, or be more widely heard.

Pleuritic friction, when acute, causes much pain, but the chronic dry pleurisy may not cause any pain. When the acute condition passes on to the effusion of serum the friction sound and the pain disappear, to reappear if the fluid is absorbed.

Simulation of crepitation sounds by friction sounds. —Sometimes a fine friction sound simulates closely a redux crepitation in character, especially when it is heard at the end of inspiration.

In such a case the effect of a cough on the sound must be tried, the crepitation being modified by it and the friction not. There is no pain with the crepitation, and there is the previous history of the case and other physical signs of changes in the spongy portion of the lung to guide the observer.

Pleuro - pericardial friction is heard over the præcordia when the subjacent pleural membrane is roughened by acute inflammation. The sound has a "to-and-fro" character, synchronous with the movements of the heart, but it may be influenced by respiration, for it sometimes disappears if the lung is fixed at the end of deep inspiration by holding the breath.

In this situation it deceptively simulates a pericardial rub, and its identification will depend on the presence of other physical signs and symptoms of pulmonary disease, and the absence of those of carditis.

VII

PHYSICAL SIGNS OF COMMON PULMONARY DISEASES

Lobar pneumonia.—***Typical.***—Onset generally acute, and often with a rigor. Herpes labialis often present.

Respiration-rate quickened out of proportion to pulse-rate.

Vocal fremitus increased. Dulness. Tubular breathing and whispering pectoriloquy. Râles and crepitations; and redux crepitation in resolution stage. Rusty sputum.

Temperature, which is high (103° to 105° F.), resolves by crisis in a few hours about the sixth day; may resolve by lysis extending over one, two, or three days.

Atypical signs are present when there is secretion in the air passages or thick lymph on the surface of the lung. Diminished or absent vocal fremitus. Dulness. Diminished or absent breath sounds.

Exploratory puncture may be necessary to diagnose between acute pneumonia and pleurisy with effusion or empyema.

The temperature may remain above normal for two or three weeks, gradually subsiding when resolution is delayed.

Acute bronchitis.—Onset more gradual.

The physical signs vary according to the severity and extent of the catarrh. Vocal fremitus is unchanged.

Rhonchial fremitus present with sonorous rhonchi. The resonance is unchanged. Wheezing is heard without a stethoscope, and rhonchi are present locally or generally, and râles if there is free secretion. Mucoid or muco-purulent sputum.

The temperature in acute bronchitis is raised to a moderate extent. In chronic bronchitis it may be normal.

Broncho-pneumonia.—Chiefly in children below five. Temperature is more elevated than in bronchitis, and lasts ten to twelve days. Physical signs vary according to the extent of the disease; may be rhonchi, râles, and impaired resonance.

Acute pleurisy.—There is a stitch pain on breathing and the face shows evidence of it. Referred shoulder pain in diaphragmatic pleurisy. Shallow respiration.

Vocal fremitus and resonance are unchanged. There is a friction rub on auscultation, heard both with inspiration and expiration, varying with the degree of inflammation. It is unchanged by cough. It passes off if effusion develops and reappears with absorption. In chronic pleurisy it may be palpable.

The temperature is raised to a moderate degree.

There is no sputum with uncomplicated pleurisy.

Pleuritic effusion.—***Small amount.***—Vocal fremitus diminished. Resonance impaired. Breath sounds distant but unchanged and not absent. No friction sound.

Larger amount.—*Typical.*—Absent vocal fremitus. Dulness. Curved line of upper limit, which does not vary with the position of the patient. Skodaic resonance. Grocco's sign. Absent breath sounds. Ægophony. Displacement of heart. Serous fluid obtained by needle.

Atypical cases.—The physical signs may be like those of acute pneumonia. Increased vocal fremitus. Dulness. Tubular breathing and whispering pectoriloquy, often distant. In these cases the fluid compresses the lung tissue, but does not obliterate the air passages.

Empyema.—More common in children than in adults. Previous symptoms of pleurisy or acute pneumonia, with temperature keeping high for an abnormally long time.

The physical signs are those of greater or less quantity of serous effusion, but more severe, with pus obtained on exploratory puncture. This is fœtid in *B. coli* affections. There may be profuse perspiration and rigors.

x-ray examination in localized or interlobar affections.

Thickened and adherent pleuræ.—Flattening of the side affected, with diminished movement. Vocal fremitus diminished, but not absent. Breath sounds diminished, but not changed. No adventitious sounds.

Atelectasis.—Airless lung with flattening of the affected side of the chest, and emphysematous enlargement of the other side. Scoliosis. Decreased vocal fremitus: dulness: absent breath sounds.

Not uncommon after pleuritic effusion, the lung failing to expand after removal of the fluid.

If the air passages remain patent the physical signs may be those of acute pneumonia. Such a condition on the left side may be the result of pressure on the lung from a large pericardial effusion.

Hydro-pneumothorax.—The fluid is pus or blood, not serous fluid.

Impaired movement of the affected half of the chest. Vocal fremitus varies according to amount of fluid: it is absent over the fluid, and diminished elsewhere. Dulness over fluid, moving with the fluid when the patient changes his position. Upper limit not curved. Succussion sounds. Coin sound over the air in the cavity. Metallic tinkling.

Phthisis.—Varies according to the extent of the lesion. The symptoms may be those of very limited bronchitis; general bronchitis; pleurisy with or without effusion; pneumonia; cavitation; fibrosis.

The temperature is of toxæmic or septic type with remissions. Tubercle bacilli in the sputum.

The previous history of the case is important, winter cough, loss of weight, early exhaustion, spitting of blood, etc.

Fibroid or interstitial pneumonia.—Chronic course. Flattening of chest over affected area, and restricted movements of chest. Vocal fremitus diminished. Impaired resonance, or dulness on percussion. Expiration is prolonged. There may be no adventitious sounds.

The heart is displaced by being pulled towards the area of shrinking.

Attacks of hæmoptysis may occur at intervals over many years, with fair health in between.

Tuberculous pneumonia.—The physical signs are like those of acute pneumonia, but with prolongation of illness, temperature remaining high, failing to resolve, and becoming toxic or septic. Tubercle bacilli in the sputum.

Cavity.—Increased vocal fremitus. Subtympanitic note. Wintrich's sign. Cracked-pot sound. Tubular

or amphoric breathing; whispering pectoriloquy. Post-tussive suction. Râles and consonating râles. Heart sounds conducted to abnormal areas of chest. Previous course of illness and symptoms. Tubercle bacilli in tuberculosis.

Sputum large in amount in bronchiectatic cavities and foul. Not foul as a rule in tuberculous cavities.

Bronchiectasis.—*Cumulative expectoration*, of a cupful or more of foul-smelling purulent sputum at intervals during the day. Signs of chronic bronchitis with cavity sometimes. Resonance may be impaired if there is much fibrosis. Heart sounds may be conducted abnormally far. Physical signs, except the large amount of expectoration, may be obscure.

Pneumothorax.—Often sudden onset with pain and dyspnœa. Enlargement of the side of the chest affected. Vocal fremitus diminished. Resonance subtympanitic or true drum note if extensive. Amphoric breathing, if pneumothorax communicates with passage into lung. Displacement of heart. Coin sound.

Miliary tuberculosis.—Continued temperature for weeks; small amount of expectoration with no tubercle bacilli as a rule; no dulness; scattered râles. There may be no physical signs except the continued septic temperature, not even any sputum.

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